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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 15 double-spaced typed pages. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

Manuscripts for and correspondence about this publication should be sent to:

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IN THIS ISSUE

CEREAL AND FORAGE CROPS

In 1955 C. G. SCHMITT, C. H. KINGSOLVER, and J. F. UNDERWOOD conducted a field test to compare development of stem rust of wheat from primary infection foci differing in concentration and arrangement, and found that rust originating from many small, distributed foci is more destructive to the crop than rust from a few large foci, page 601.

S. M. PADY and C. O. JOHNSTON's cereal rust summary for the 1958 season in Kansas stresses the importance of stripe rust of wheat; damage from stripe rust was greater than the combined loss from leaf and stem rust, page 607.

A more flexible, but still uniform, system of wheat leaf rust race identification and nomenclature which would retain the standard differentials and supplement them with other suitable differential and universally resistant varieties is proposed by a COMMITTEE OF NORTH AMERICAN WHEAT LEAF RUST RESEARCH WORKERS, page 613.

WILLARD F. CROSIER reports results of seed treatment trials with several antibiotics for control of grain smuts, page 616.

Of the 24 varieties of alfalfa tested by CHARLES M. LEACH and JOHN R. HARDISON in field trials in western Oregon for susceptibility to crown wart, none was resistant, but differences between varieties were significant, page 619.

According to CHARLES M. LEACH, who conducted a survey of root deterioration of alfalfa in Oregon, both vascular discoloration and insect injury are more important than cortical injury in contributing to the decline in yields of alfalfa plantings, page 622.

VEGETABLES (see also under Virus)

D. M. HUBER and A. M. FINLEY have demonstrated, for the first time, the pathogenicity to bean roots of Gliocladium roseum, an organism previously thought to be unimportant in the bean root rot complex, although it is known to be a common inhabitant of soils in Idaho, page 626.

A 3-year study in Idaho on the incidence of spotting of lettuce cotyledons has led A. M. FINLEY to the conclusion that spotting is related directly to the type of season and is a manifestation of physiologic drought, while differences in susceptibility between varieties are unimportant, page 629.

In the course of a continuing investigation being undertaken in Michigan for an effective control of potato scab, H. S. POTTER, W. J. HOOKER, W. CARGO, and G. T. STACHWICK tested PCNB and urea-formaldehyde, and from their results they consider that these materials are potentially useful in potato production, page 633.

VIRUS

C. E. YARWOOD reviews evidence supporting the hypothesis that some plant viruses evolved in roots, as well as a comparable hypothesis for the origin of other plant viruses in their insect vectors, pointing out the similarities between the two points of view and noting that they are not at all mutually exclusive, page 638.

The first report of the natural occurrence in the United States of a systemic infection of bean caused by a tobacco necrosis virus is given by JOHN J. NATTI from New York, page 640.

Most aster yellows-infected strawberry nursery plants die soon after planting out in California, according to a study made by NORMAN W. FRAZIER and HAROLD E. THOMAS, page 645.

FRUITS AND NUTS (see also under Virus)

Results of experiments by P. M. MILLER and E. M. STODDARD indicate that thiram is superior to dichlone and captan in reduction of pre- and post-harvest rot of strawberries, and that lengthening of the time between the last spray and harvest increases severity of grey mold, page 646.

During a study to investigate the development of a red variant of the blue mold fungus on lemons stored in presence of biphenyl, PAUL R. HARDING, Jr. obtained resistant and semi-resistant strains of the fungus, produced by continued exposure to biphenyl, page 649.

JACK TAYLOR describes three important rots of apples in Georgia and compares symp-

toms, severity, losses, and other features with the same diseases as found in other eastern apple areas, page 654.

JOHN R. COLE emphasizes the importance of following the spray schedule recommended by Crops Research Division, Agricultural Research Service, to control scab on the Schley variety of pecan in central Georgia, page 658.

TECHNIQUES

A method for making rapid photomicrographs that employs high-contrast film is suggested by WILLIAM J. STONE and JOHN P. JONES, page 659.

In greenhouse tests over a 3-year period, chemical 5400 was the best of numerous materials tried by O. D. MORGAN for control of algae, mosses and fungi producing a nuisance growth on greenhouse benches, pots and soil, page 660.

MISCELLANEOUS

D. P. TAYLOR's discovery of *Meloidogyne javanica* on Jerusalem cherry constitutes a first report of this species of nematode on Jerusalem cherry as well as the first report of root knot on this host in a Minnesota greenhouse, page 664.

From his experiments on the relationship between dirting and non-dirting and the role of dinoseb in peanut stem rot control, KENNETH H. GARREN has concluded that use of dinoseb in non-dirting cultivation can be justified only on the basis of weed control and not on its fungicidal activity, page 665.

A severe outbreak of a leafspot disease, caused by *Cercospora theae*, occurred on several thousand plants of two varieties of *Camellia sasanqua* and on four *Thea sinensis* plants growing in lath houses in two different Louisiana nurseries, according to A. G. PLAKIDAS, page 668.

H. K. SAKSENA and B. B. SINGH have identified the causal organism of a new blight of marigold in India as the same fungus that causes dieback of chillies, page 670.

Brief Note on Plant Disease Occurrence, page 673: Tar spot of corn in Guatemala, by EUGENIO SCHIEBER.

April Weather, page 674.

EPIDEMIOLOGY OF STEM RUST OF WHEAT: I. WHEAT STEM RUST DEVELOPMENT FROM INOCULATION FOCI OF DIFFERENT CONCENTRATION AND SPATIAL ARRANGEMENT

C. G. Schmitt[1], C. H. Kingsolver[2] and J. F. Underwood[3,4]

Summary

Development of stem rust of wheat (*Puccinia graminis tritici* Eriks. & E. Henn.) was studied in a field of Thorne wheat from infection foci of initial intensities of 30 and 900 leaf pustules per foot of row. Increase in disease was most rapid in the plot in which 900 pustules were distributed within a circle of 20-foot radius, compared with the same number of pustules concentrated in a single lineal foot of row. The least rapid increase was in the plot in which 30 pustules per lineal foot of row served as the initial focus for infection. The disease increase was not of the same order of magnitude as the difference in the number of pustules used in the initial inoculation. The data indicate that many small infection foci distributed over a field are more destructive to the crop than are a few large foci. Coefficients of regression for rust intensification ranged between $b_e = 0.252$ and $b_e = 0.417$.

cf. 38, 195

INTRODUCTION

A field test was conducted at Fort Detrick during the spring and summer of 1955 to compare rust development on wheat from primary infection foci. Plants for establishing foci were grown in the field, removed to the laboratory, inoculated, incubated under controlled conditions, hardened in the cold frame, and finally planted in the field. This method of initiating disease in the test was chosen to achieve more precise control over primary infection strength than seemed possible either by dusting talc-spore mixtures on plants or by hypodermic injections of aqueous suspensions of uredospores into plants.

EXPERIMENTAL PROCEDURES

Three circular plots, each 400 feet in diameter, were established in a field of Thorne winter wheat. The field was sown on October 6, 1954 at the rate of 76 pounds per acre in rows spaced 7 inches apart. About 380 pounds of 5-10-5 fertilizer were applied per acre at seeding. On April 8, 12-inch sections of crop row were dug, replanted in flats and taken into the laboratory for immediate inoculation with race 56. Following inoculation, the flats were placed first in a dew chamber for 16 hours and then transferred to a greenhouse held at 68° to 75° F for the first 5 days of the rust incubation period. The plants were then hardened in a cold frame until pustules were present (April 20). On this date the foci were established in the 400-foot circular plots by replanting selected crop row units which had been carefully examined and trimmed to contain the desired numbers of young pustules. The three centers of infection formed were of the following composition:

Plot W-1: Twelve inches of crop row, containing 30 pustules, in the center of the plot.

Plot W-2: Twelve inches of crop row, containing 900 pustules, in the center of the plot.

Plot W-3: Thirty 12-inch crop row units, each containing 30 pustules, placed at points as equidistantly spaced as possible in a 6-row, 5-column pattern within a circle of 20-foot radius from plot center.

In each of the three plots 24 radii were projected outward from the center at 15 degree intervals. To designate observation points, 3/4-inch x 3/4-inch wooden stakes 5 feet long

1Plant Pathologist, Biological Laboratories, U. S. Army CmlC, Ft. Detrick, Frederick, Maryland.
2Quality Maintenance and Control Section, AMS, Beltsville, Maryland.
3Southern States Cooperative, Upper Marlboro, Maryland.

4The authors wish to express their appreciation to Thomas R. Tegenkamp, Gerald A. Jung and Edward J. Ryder for technical assistance.

were vertically positioned on each radius at 20, 40, 60, 80, 100, 120, 140, and 160 feet from plot center. In each plot the established observation points thus formed a polar coordinate pattern consisting of eight concentric circles intersecting 24 radial lines. Supplemental data were also obtained from locations on intermediate unmarked rings between these fixed observation points at intervals during the experiment.

Pustules were counted on units of 20 culms of wheat near the staked observation stations until intensification of rust made counting impractical. When this stage was reached, severity was estimated in percentage values by referring to the modified Cobb scale. First observations were made on May 2 and repeated thereafter at approximately 2 to 4 day intervals until crop maturity terminated further disease development. The last observations were on June 21.

Weather instruments of three types were utilized in or near the test field. A hygrothermograph in a louvered wooden shelter was placed in the wheat field on the ground among the plants to record temperature and relative humidity. A Taylor dew meter[5], also placed near the ground, measured dew deposition. An AN/GMQ-1 modified recording anemometer[6] continuously recorded the directional and speed characteristics of the winds at a 5-foot height above the ground. The wind recording device was located along one edge of the field in a position free from obstructions to wind flow.

RESULTS AND DISCUSSION

In some wheat farming areas crop losses because of naturally occurring wheat stem rust are sometimes negligible. In these instances inblown inoculum may arrive too late in the growing season to cause serious damage. The present experiments indicate that under similar conditions extensive crop damage could occur by harvest time if even extremely modest amounts of inoculum arrived early enough in the growing season. In the latter stages of the experiment a light rust infection from naturally occurring spore showers was observed throughout Frederick County. The inblown natural inoculum arrived so late, however, that its influence on the yield of the commercial wheat crop was negligible.

First evidence of secondary infection around introduced foci was observed May 2, 13 days after rusted plants were transplanted into the field. Most of the pustules were found within 2 feet of the original focus. No rust was observed on this date in the ring of observation stakes 20 feet from plot center or beyond. Even in plot W-3 no rust was found beyond the boundary of the circle in which the original foci were established.

From May 2 to 23 nearly all obvious secondary infection of stem rust was confined to an area within a radius of about 20 feet from each focus in plots W-1 and W-2 and within a radius of about 30 to 40 feet in W-3. Even though some of the artificially infected plants were located near the periphery of the 20-foot circle in plot W-3, the extent of rust development was comparable in all three plots.

Earliest disease development was found toward the southwest, the predominant direction of wind movement for the previous 2 weeks. Subsequently stem rust developed irrespective of wind direction and was found in all portions of the field by mid-May. Disease development proceeded more rapidly after mid-May when the incubation period shortened with warmer temperatures, from 13 to 9 days.

Successive observations after May 23 discolosed rapid rust development. By June 6 plants within 100 or 120 feet of all three foci exhibited sufficient infection to be expressed in severity percentages. Plants within 20 to 30 feet of the foci were being killed by stem rust. Terminal readings were made on June 21 when the crop was nearly mature. Minimum severity was 20 to 25 percent.

Scale diagrams are presented to compare the progress of rust spread from the three infection foci (Figures 1, 2 and 3). Isopleths denote areas that attained a 0.5 pustule per culm or greater level of infection. The numbers between isopleths expressed elapsed time in days since the date spores were first liberated by the foci. Day zero was April 20, the date that diseased plants were replanted in the field. A few of the pustules were liberating spores at this time, but the majority did not actively release spores until 2 days later.

A roughly elliptical pattern of outmovement was obtained from this test. While the focus

[5]Taylor, Carlton F. 1956. A device for recording the duration of dew deposits. Plant Disease Reptr. 40: 1025-1028.

[6]This consists of a mast and wind transmitter as supplied by the Army Signal Corps modified by Ft. Detrick personnel with a specially constructed bridge and rectifier circuit controlling two Esterline Angus recording milliammeters.

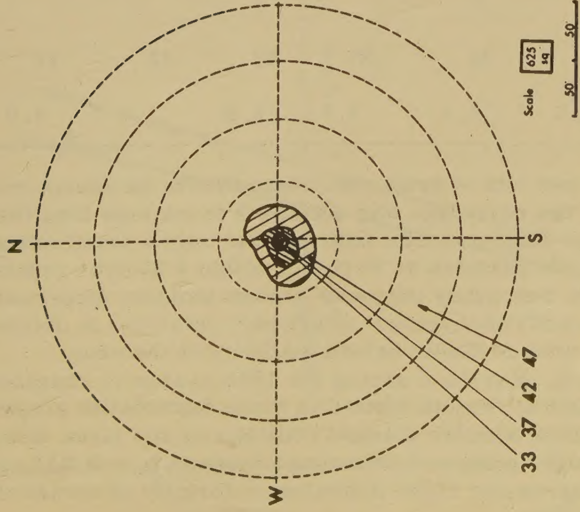

Figure 1. Time required for rust spread from the 30-pustule concentrated focus to a 0.5 pustule per culm level. Numbers represent days since infected plants were placed in the field.

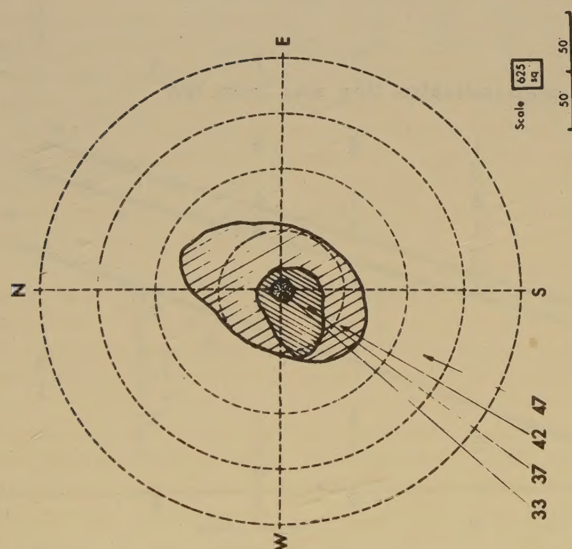

Figure 2. Time required for rust spread from the 900-pustule concentrated focus to a 0.5 pustule per culm level. Numbers represent days since infected plants were placed in the field.

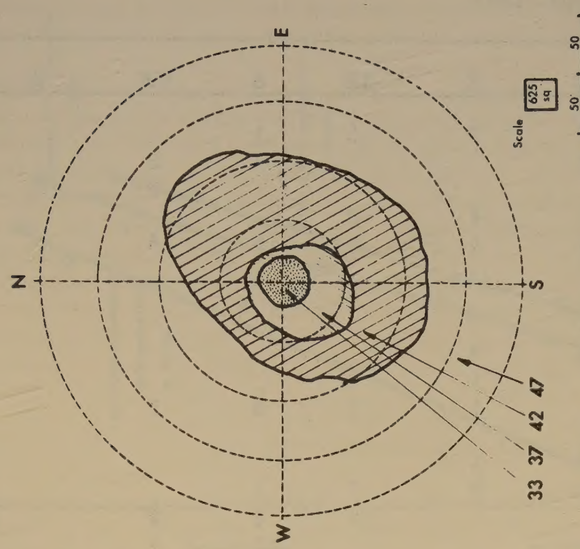

Figure 3. Time required for rust spread from the 900- pustule dispersed focus to a 0.5 pustule per culm level. Numbers represent days since infected plants were placed in the field.

Table 1. Frequency of mean hourly direction of daytime winds (0700-1900 hours inclusive) by days, Frederick, Maryland, 1955.

Date	Calm	N	NE	E	SE	S	SW	W	NW
April 20		1	4	3	3	1			
21		4	2			3	3		
22							2	5	5
23				4	8				
24			3	1	1	3	4		
25		5	7						
26	2	1	9						
27		4	8						
28	1		8	3					
29		9		1					2
30		4	1	1	1	3	1		1
May 1		1	6	4					1
2		1	1	2	7	1			
3	5				2	3	2		
4						11	1		
5		1						1	10
6		1						1	10
7					1	7	4		
8	Data missing but synoptic map indicates flow was from NW								
9		4							8
10		1	1	1	5	3			1
11	3	2	4	3					
12	2		1	2	1	6			
13			2	1	1	4			
14	2	2	6	2					
15	2	2	5	1	1	1			
16						7	5		
17		1	11						
18								3	9
19		2						1	9
20	1		3		2	3	3		
21	1		6	2	1	2			
22	6					6			
23			4		3	4	1		
24			6	6					
25						1	1	6	4
26		3	6	2		1			
Total hours	25	47	104	39	37	69	32	17	60
% Wind	5.8	10.9	24.2	9.1	8.6	16.0	7.4	4.0	14.0

originally comprising 900 pustules per foot of crop row induced more extensive rust development than did the 30-pustule focus, the advantage was definitely much less than the 30-fold difference in original strength. The diagrams also indicate that only a small advantage was achieved by slightly dispersing the 900 pustules at 30 points within a 20-foot radius. Results indicate that maximum damage from secondary increase follows uniform dispersal of primary infection. This presupposes that a sufficient amount of primary infection is obtained to intensify to destructive levels in the time available before maturity of the crop.

Rust intensification at Frederick, Maryland during the 1955 season is characterized by regression lines in Figures 4, 5, and 6. Points plotted in these exponential graphs are means of several readings from areas of quite similar disease reaction on any given date. Coefficients of regression values of the eight groups of data range between $b_e = 0.252$ and $b_e = 0.417$. The closeness of fit to the linear regression slope indicates uniformity of measurement by pustule count and severity estimate data.

The 1955 season was favorable for rust development. Dew duration was seldom the lim-

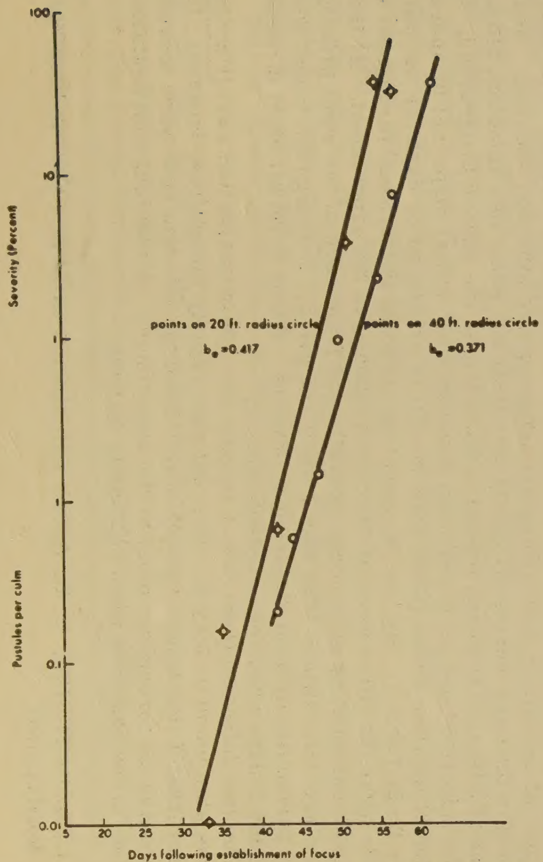

Figure 4. INTENSIFICATION OF RUST AROUND 30- PUSTULE FOCUS (Plot W-1), 1955

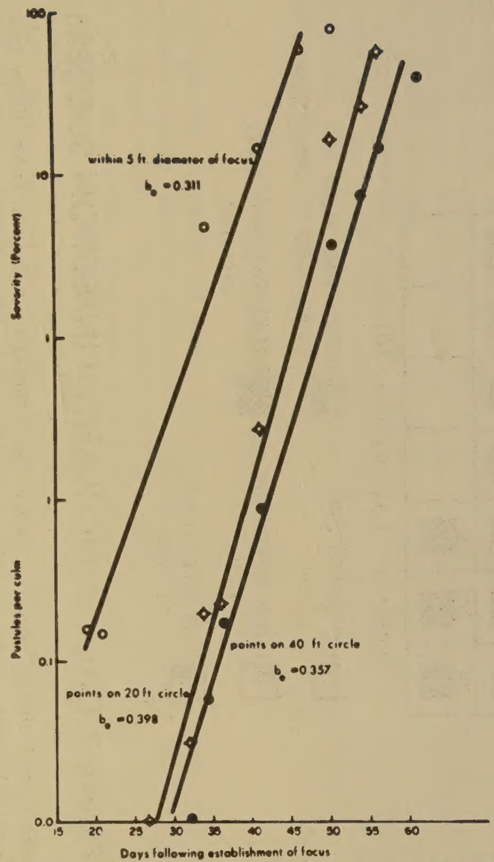

Figure 5. INTENSIFICATION OF RUST AROUND 900-PUSTULE CONCENTRATED FOCUS (Plot W-2), 1955

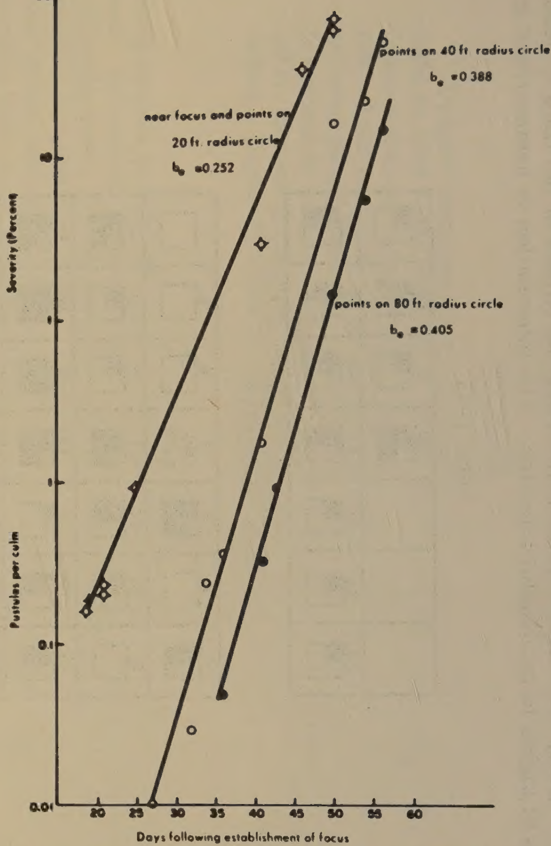

Figure 6. INTENSIFICATION OF RUST AROUND 900-PUSTULE DISPERSED FOCUS (Plot W-3)

iting factor, for there were only 7 nights during the period April 20-May 31 with less than 5 to 6 hours of dew. Daily inoculations were made along the border of the field to determine the conduciveness of nights to successful infection. The information is summarized in Figure 7.

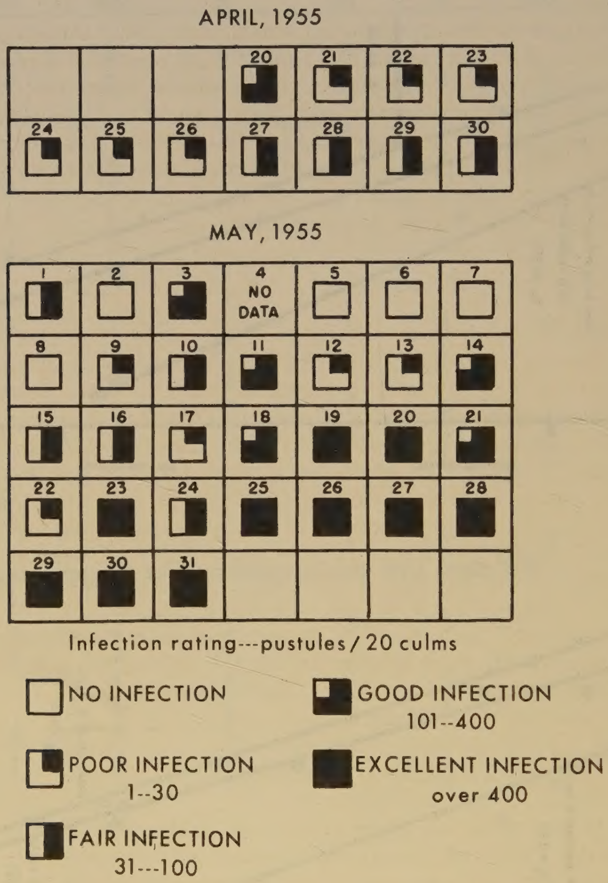

Figure 7. DAILY INOCULATION INFECTION SUCCESS

The evaluation is based on the relative numbers of pustules obtained from daily inoculations made under a portable settling tower with equivalent amounts of inoculum. The data largely reflect the impact of prevailing meteorological conditions on infection success. Nearly all nights in the period involved were sufficiently favorable for some new infection although the relative amount of infection achieved tended to increase as the season progressed. The four nights rated "no infection" did not occur until 3 weeks after initial dispersal of spores.

Distribution of daytime winds (0700-1900 hours) is presented by days for the period April 20 through May 26 in Table 1. By late May rust had become established throughout the field. Therefore winds after that time were of little consequence in the experiment. At the bottom of Table 1 values representing percentage overall wind from each of the eight principal directions and percentage calm (winds less than 2 mph) are given. Although northeast winds predominated, the variability of wind direction is obvious. Winds from all eight directions were represented within 3 days after focus establishment.

A diffuse natural infection (less than 1 pustule per 100 culms) of leaf rust (*Puccinia rubigo-vera tritici*) was also found on May 2. The leaf rust rapidly intensified, however, to cause almost complete loss of leaf tissue by mid-May. Readings of stem rust were therefore restricted to stem tissues throughout most of the experiment. A few leaf rust pustules developed on stems but they were excluded when recording disease.

BIOLOGICAL LABORATORIES, U. S. ARMY CHEMICAL CORPS, FORT DETRICK, FREDERICK, MARYLAND

CEREAL RUST AEROBIOLOGY AND EPIDEMIOLOGY IN KANSAS IN 1958[1]

S. M. Pady and C. O. Johnston[2]

Summary

Despite favorable growing conditions, rust infections were light in 1958 so far as leaf and stem rust of wheat were concerned. Spore showers were frequent, but numbers of spores were very low. Leaf rust caused an estimated loss of 0.5 percent; stem rust, no loss; stripe rust, appearing for the second straight year, was prevalent in western Kansas and caused an estimated 1 percent loss. Losses from rusts on other cereals, except leaf rust of rye (about 1 percent), were too low to be estimated.

The growing season of 1957-58 was favorable for wheat, and rust damage was the lowest for many years. An unusual feature of the 1958 rust picture was the prevalence of stripe rust, Puccinia striiformis West. (P. glumarum (Schmidt) Eriks. & E. Henn.), in the western half of Kansas, causing an estimated loss of 1 percent. This was the second time in the history of the State that stripe rust had appeared, and for the second straight year. The first time it caused appreciable loss; in fact, damage from stripe rust was greater than the combined loss from leaf rust (P. recondita Rob. ex Desm. f. sp. tritici) and stem rust (P. graminis Pers. f. sp. tritici Eriks. & E. Henn.), estimated at 0.5 and 0 percent, respectively. An account of the stripe rust development in Kansas in 1958 has been published separately (5).

The growing season, from the fall of 1957 on, was unusually favorable, with abundant rainfall and no drouth periods. As a result, a record crop of 291,252,000 bushels from 10,591,000 acres (an average yield of 27.5 bushels per acre) indicates an unusually favorable year. As in the past (1, 2, 3), rust spores in the air during the fall, their numbers during the growing season, and their development in the field were observed. Meteorological conditions also were observed, as in the past.

FALL AND WINTER CONDITIONS

Sufficient rain fell in nearly all parts of Kansas during the summer and fall of 1957, making conditions nearly ideal for preparing wheat seedbeds, seeding, and obtaining good stands of well-rooted wheat. The supply of soil moisture was the best in the western two-thirds of the State since 1940. Moisture penetrated to 45 inches or more, and an average of 7.78 inches of moisture had been stored in the upper 4 feet by April 1. Average moisture content of the soil in the western two-thirds of the State was 19.5 percent, well above the 16.9 percent in 1957. Soil moisture and weather conditions were so favorable for seeding the 1958 wheat crop that excellent stands were obtained. Tillering and root development were excellent, and good growth was made in the fall.

Spore traps were exposed daily at the sampling station on the roof of Willard Hall, Kansas State College campus, September 24 to November 1. Silicone-coated microscope slides were exposed for 24 hours or longer in a windvane-type holder which keeps the slides at a 45° angle facing the wind. The numbers of leaf and stem rust spores trapped, along with applicable meteorological data, are recorded in Table 1.

Rust inoculum in general was not abundant; leaf rust occurred on only four slides and stem rust on six, and in all cases numbers of spores were low. It should be noted that stem rust spores were present in the air September 24, following 4 days of north and northwest winds. Northerly winds October 7-10, 16, 23, and 24 carried either leaf or stem rust spores (Table 1). This is further evidence that northerly winds carry urediospores southward during October.

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[1]Contribution No. 529, serial No. 681, Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, Manhattan.

[2]Respectively, Head, Department of Botany and Plant Pathology, Kansas State College, and Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, located at Kansas State College.

Table 1. Numbers of stem rust and leaf rust spores trapped on silicone-coated slides exposed on roof of Willard Hall, Kansas State College campus, calculated on a square-foot basis, September 24 to November 1, 1957.

Date	Number of spores per square foot		Precipitation (inches)	Wind		Temperature (°F)	
	Leaf rust	Stem rust		Direction	m/p/d movement	Maximum	Minimum
Sept. 24	----	1650	----	S	156	86	45
25	----	750	----	S	132	81	52
26	----	----	----	E	65	78	48
27	----	T	----	E	143	74	51
28, 29	T	----	----	E, E	107, 70	76, 79	45, 41
30	1065	----	----	S	52	84	40
Oct. 1	----	----	----	E	99	84	48
2	----	----	----	E	101	81	44
3	----	----	----	E	181	81	51
4	----	1065	----	E	214	81	51
5, 6	----	----	T	E, S	200, 175	79, 84	56, 54
7, 8	6390	----	T, 2.23	S, N	107, 210	74, 62	61, 54
9, 10	T	----	T, ----	NE, E	198, 111	56, 59	49, 44
11, 12, 13	----	----	T, .03	E, NE, SE	149, 156, 165	64, 70, 64	47, 51, 50
14	----	----	.02	SE	215	65	54
15	----	----	.23	S	71	67	58
16	----	710	T	N	101	70	49
17	----	----	----	N	136	63	40
18	----	----	----	E	42	64	33
19, 20	----	----	----	E, E	94, 116	63, 64	35, 35
21, 22	----	----	T, .44	S, S	143, 136	60, 66	46, 54
23, 24	----	T	.45, .10	NW, N	199, 236	63, 50	50, 40
25, 26, 27	----	----	T, --, T	N, N, S	181, 58, 79	41, 32, 48	28, 22, 23
28	----	----	----	S	212	62	26
29	----	----	----	W	119	64	40
30	----	----	----	NW	98	73	39
31	----	----	----	E	113	70	34
Nov. 1	----	----	----	S	220	71	46

Despite the good stands of wheat and the lush growth in some localities, there was little fall infection by cereal rusts. This was partly due to the absence of volunteer wheat in large sections of the State. For example, there was little wheat grown in the area south of Goodland and west of Dodge City in 1957. Therefore there was very little volunteer in western Kansas. This delayed the initiation of fall infections, which in turn resulted in lighter infections than usual. Elsewhere in the western half of Kansas volunteer wheat was confined mostly to roadsides, ditch banks, and swales in occasional fields. Consequently there was little on which the rusts of wheat could develop. As a result, there was very little fall infection. On November 1 leaf rust was observed on volunteer wheat in all fields visited in a field trip through seven central Kansas counties. However, infection was low in most fields. Stem rust was found in only one field.

The winter was mild with only a few short periods of near-zero weather. Rain or snow fell frequently throughout the winter and spring. Extremely heavy snow fell in the northwestern fourth of the State during February and March and remained on the ground for long periods. Each time there was severly cold weather there was an ample cover of snow to protect wheat. As a consequence, winter wheat came through the winter in excellent condition in all parts of the State.

On March 20 a few uredia of leaf rust were found overwintering on volunteer wheat growing in a protected spot near Manhattan. No overwintering of cereal rust was observed else-

where, nor were any reports of its occurrence received. The small amount of leaf rust present in some fields disappeared during the winter, and no rust could be found on winter wheat as it came from dormancy in April.

SPRING INFECTIONS AND AIRBORNE SPORES

Leaf rust infections began increasing in Oklahoma during late January and early February, but a severe cold spell February 9 to 13 killed many wheat leaves and leaf rust infections there were greatly reduced.

Spring was late and very cool in Kansas, with frequent light rains and snows. Although temperatures were excellent for growth of winter wheat, they were too low for early and abundant development of rust. The first spring infections of leaf rust were observed near Winfield on April 28. From that date on, the development of leaf rust was very slow. A month later there were only traces of leaf rust in many fields.

Daily exposures of silicone-coated slides were started on March 25 and continued through July 29 to determine the numbers of spores in the air and the frequency of spore showers. In Figure 1 the numbers of rust spores, meteorological data on precipitation, wind direction and movement, maximum and minimum temperatures, and infection periods are recorded for April 25 through July. Because of negative readings on the rust slides, data are presented for the period beginning April 25, rather than March 25.

LEAF RUST OF WHEAT

The first infections of leaf rust in the field were found April 28 in four south-central counties bordering Oklahoma. This was approximately the same time that the disease appeared in 1957 and is about average for its appearance in the State (4). An interesting observation was that there were no spore showers recorded at Manhattan prior to the appearance of the rust in the field. Since Manhattan is about 140 miles north of the first sites of infection, it is probable that the spores that initiated those infections did not reach so far north as Manhattan.

The first spore showers occurred May 20-24, although traces were present May 6 and 7 (Fig. 1). Infection periods are shown in Figure 1. These were periods when favorable moisture, southerly winds, and abundant spores in the air coincided. Favorable periods were present at the end of May and occurred at frequent intervals throughout June. Rain fell on 14 days with a total of 7.94 inches an excess of 2.85 inches. Southerly winds occurred on only 12 days, but on only two occasions did a southerly wind blow on successive days, June 3-4 and 27-30. This may help to explain why the amount of inoculum was so low in May and why development of rust in the field was retarded.

The heaviest spore showers occurred May 30 and June 3, but the peaks were only 130,285 and 115,370 leaf rust spores per square foot, compared with the peaks of 1957, which were three to four times higher. By the end of May, leaf rust still was present in only trace amounts in many fields in the State.

Favorable infection periods and small spore showers continued during the first half of June so that by the end of the month leaf rust was moderately heavy in occasional fields of susceptible varieties. However, leaf rust was very light for the State as a whole, and is estimated to have reduced the average yield by only 0.5 percent. This is strikingly different from the 1957 season, when leaf rust caused an estimated 10 percent loss. That 1958 was a light rust year is indicated by the fact that during the last 20 years in Kansas leaf rust losses have been higher in 15 of those years than they were in 1958. Other light leaf rust years were 1956, 1953, 1950, 1948, 1947, and 1940, when losses were trace, 0, trace, 0, trace, and trace, respectively (4).

STRIPE RUST OF WHEAT

Stripe rust, which was present in Kansas for the second straight year (6), was the most conspicuous rust in the State in 1958 (5). Traces were first found in Chase, Marion, Reno, and Saline counties May 12. From then on through May and the first half of June there was a gradual buildup of infection, especially in the western half of the State. Susceptible varieties had stripe rust from 20 to 50 percent severity by June 1. This caused severe leaf drying on highly susceptible Wichita, Bison, Concho, and Kiowa in many fields.

The urediospores of stripe rust are usually indistinguishable from those of leaf rust under ordinary magnification; so it is highly probable that most of the spores included in leaf rust in

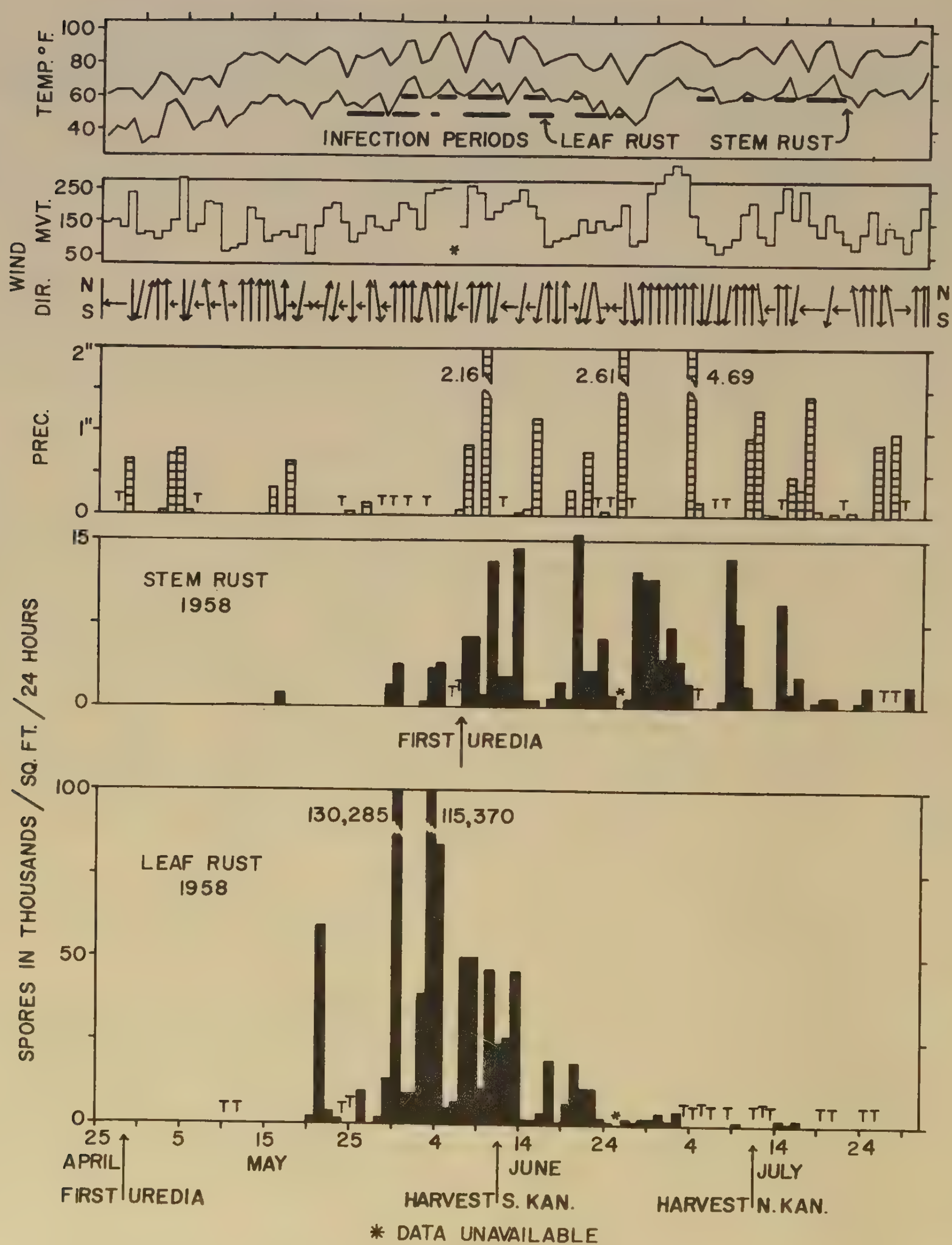

FIGURE 1. Numbers of rust spores trapped per square foot per day on silicone-coated slides exposed on the roof of Willard Hall, Kansas State College campus, from April 25 through July 31, 1958. Meteorological data supplied by campus station. Leaf rust column includes stripe rust spores.

Figure 1 are those of stripe rust rather than of leaf rust. This is suggested also by the unusual circumstance of finding stripe rust in abundance in fields while leaf rust was scarce. Observations made in central and western Kansas May 26 and 27 revealed that stripe rust was present in every field examined in 14 counties. In the Manhattan area only a trace was present, but from Salina west prevalence was 100 percent with severity up to 50 percent in some fields. There was less stripe rust from Garden City west. Leaf rust was present also in practically every field with prevalence of a trace to 100 percent, but severity in all cases was low. Hot weather in early June caused drying of the stripe rust-infected foliage, but this was offset by favorable moisture conditions and stripe rust did not cause the damage feared earlier. Nonetheless, stripe rust caused extensive damage to wheat in western Kansas, and the loss was estimated to be 1.0 percent for the State as a whole (5).

The unusual situation of stripe rust causing a 1 percent loss in Kansas, while leaf and stem rust combined caused a total of 0.5 percent loss, is probably the outstanding feature of the 1958 rust season. Appearing in 1957 for the first time in the history of the State, stripe rust's presence in 1958 in damaging amounts raises the question of whether stripe rust should not be considered as one of the important wheat rusts of the Plains area.

STEM RUST OF WHEAT

Stem rust was late in appearing in the State, the first pustules being found June 6, which is about a week later than the average date (4). Stem rust spores were present in the air in small numbers May 16, 29, and 30. This coincided with favorable infection periods (Fig. 1) and probably accounted for the first field infections. Although abundant moisture and numerous infection periods occurred during the first half of June, stem rust was very slow to develop and infections were light and scattered.

Numbers of stem rust spores in the air during June 1958 were very low as compared with the 1957 season, being only one-tenth as many. The maximum number of spores trapped was 152,000 per square foot, compared with the maximum of 465,760 in 1957. From June 20 to July 4 temperatures were unfavorable for further spread and development of cereal rusts. This condition, in combination with light inoculum, resulted in the confining of stem-rust infection to low spots in occasional fields, and its occurrence as only a trace in other fields. By harvest time it was evident that stem-rust damage would be extremely light, and it was estimated that it did not cause any measurable loss. This is only the third year since 1932 that there has not been some loss from stem rust; zero losses were recorded for only 1942 and 1946.

OTHER CEREAL RUSTS

Both crown rust and stem rust were late in their inception on oats in Kansas in 1958. Infections increased only very slowly during the cool, rainy weather of June. By harvest time traces of both rusts could be found on susceptible varieties, but it was estimated that the amount was insufficient to cause measurable reductions in yield.

Considerable leaf rust of barley developed in experimental sowings of winter and spring barley at Manhattan during May, but it disappeared as temperatures increased and could not be found in June. Infections were mostly confined to the lower leaves. No leaf rust was seen in commercial fields of winter barley and only occasional pustules of stem rust. It was estimated that neither leaf rust nor stem rust caused a measurable loss in the yield of barley in 1958.

Leaf rust of rye was fairly abundant but not particularly destructive. The acreage of rye in Kansas was higher than usual in 1958; so yields probably were reduced by leaf rust. This was estimated to be about 1 percent. Traces of stem rust were present on rye, but it is believed that it did not cause a measurable loss in yield.

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, KANSAS STATE COLLEGE, MANHATTAN, IN COOPERATION WITH CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

A PROPOSED MODIFICATION OF THE SYSTEM OF WHEAT LEAF RUST RACE IDENTIFICATION AND NOMENCLATURE

A Committee of North American Wheat Leaf Rust Research Workers[1]

A dynamic, open-end system of wheat leaf rust (Puccinia recondita Rob. ex Desm.) race identification and nomenclature is needed which will serve in studies of the classification and distribution of new or previously undetected factors for virulence and yet retain continuity with past and present epidemiological studies and systematics. Such a system should involve the use of supplemental differential and resistant wheat varieties. The use of additional or supplemental varieties for the identification of certain types of virulence has been suggested before.

Waterhouse (8), in 1932, proposed the use of a supplemental differential for wheat leaf rust, and Vallega (6), Vallega and Favret (7), da Silva (4), and possibly others have since suggested that supplemental differentials would be useful. Loegering and Stakman (3) and Johnson and Green (1) have proposed the use of supplemental differentials for wheat stem rust also, and Stakman and Stewart[2] proposed a method for selecting supplemental differentials and a system of nomenclature for stem rust associated with the use of supplemental differentials. In these proposals the standard differentials were retained.

Simons and Murphy (5), on the other hand, have developed a new independent set of differentials for oat crown rust. This set retains some of the old differentials together with some new varieties and is used with a new key and race numbers. While the use of supplemental or new differentials has furnished valuable information for plant pathologists and plant breeders, their use in wheat leaf rust race identification has not yet been systematized.

The general problems concerned with the use of supplemental differentials in leaf rust race identification and the attendant nomenclature were discussed at a meeting at Stillwater, Oklahoma, in February 1958, and again at Winnipeg, Manitoba, in August of that year. It was concluded that the establishment of a new set of differentials with attendant new keys and race numbers would eventually create the same shortcomings encountered with the standard differentials now in use. The system herein proposed would, therefore, retain the standard differentials and would supplement these with other suitable differential and universally resistant varieties. It was agreed to adopt the methods proposed by Stakman and Stewart[2] for the selection of supplemental differentials but to try on an experimental basis a different system of nomenclature to accommodate the use of supplemental differentials.

Thus there will be three categories of varieties in use in this program:

1. Test varieties. (Varieties being tested as possible supplemental differentials.)
2. Supplemental differentials. (Varieties selected from the "Test varieties" which are useful in differentiating leaf rust virulences not detected by the standard differentials and also varieties which are currently resistant to all leaf rust cultures.
3. Standard differentials. (Varieties in the set of differentials published by Johnston and Mains (2) in 1932.)

It was further proposed that an attempt be made to establish a uniform group or set of supplemental differentials for use at least throughout North America. Such a set of supplemental differentials will be established only after adequate experience with the test varieties. The varieties suggested for initial testing are listed in Table 1. It is emphasized that this is not a list or set of supplemental differentials, but only a list of varieties that will be tested for possible inclusion in a set of supplemental differentials to be established at a later date. By cooperative agreement, the supplemental differentials so selected may be changed from time to time as the need arises. Additions to the supplemental set will always be made from a list of test varieties that will be under study continually.

A system of nomenclature to be used when the supplemental differentials are used in leaf rust race identification was agreed upon. The name, or designation, of a race will consist of

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[1] W. Q. Loegering, Chm., U.S.D.A., Beltsville, Md.; C. O. Johnston, U.S.D.A., Manhattan, Kans.; D. J. Samborski, Univ. of Manitoba, Winnipeg, Man. Can.; R. M. Caldwell and J. F. Schafer, Purdue Univ., Lafayette, Ind.; H. C. Young, Jr., Sec., Okla. State Univ., Stillwater, Okla.

[2] Stakman, E. C., and D. M. Stewart. 1957. Taxonomy of Physiologic Races of Puccinia graminis var. tritici. U.S. Dept. of Agr., Agr. Res. Service, Plant Pest Control Div., Coop. Rust Lab. Memorandum. April 10, 1957. Processed.

Table 1. Wheat varieties under test as possible supplemental differentials for wheat leaf rust race identification, January 1959.

Name	C. I. Number	Selection Number
1. Agrus	13228	Purdue 39120A5-3-1-1-1-3
2. Newsar	12530	Purdue 3848A5-5-1-36
3. Waban	12992	Purdue 3369-61-1-1-10R
4. (Honor^2 - Rosen Rye x Yorkwin) x Cornell 595	13078	Cornell 82 a1-2-4-7
5. Wardal	13372	Purdue 4665A2-9-1
6. Sinvalocho	12595	D. I. V. 8385
7. Klein Lucero		D. I. V. 8386
8. Klein Titan		D. I. V. 396
9. Westar	12110	
10. Wesel	13090	
11. Exchange	12635	
12. Rio Negro	12469	
13. Colotana 266/51	13556	P. I. 214392
14. Lee	12488	
15. Aniversario	12578	
16. Transfer, Chinese + *Aegilops umbellulata*	13483	P54-47.4-6

three parts which will be separated by hyphens. The first part will be a race number determined on the basis of the reaction of the eight standard differentials. The second part will be a designation for the specific set of supplemental differentials. This designation will consist of letters indicating the area of acceptance of the supplemental differential set and a two-digit number indicating the year in which the list of varieties included in the specific supplemental differential set was published. The designation for the uniform set established for North America will be "NA." A local area designation, such as "Okla" is to be used when any local worker establishes a supplemental differential set for his own use that is different from the "NA" set used throughout North America. The list of varieties for the "NA" set of supplemental differentials will be published in the "Plant Disease Reporter" and in "Robigo." No supplemental differential set will be recognized for purposes of nomenclature until the list of varieties in it has been published. The third part of the designation will be a number of a consecutive series used to indicate cultures which produce certain specific reactions on the supplemental set of differentials. The numbers of this consecutive series will be assigned in order of recognition of pathogenically different cultures. Where the "NA" supplemental differential set is used, it will be important that there be a certain individual responsible for assigning numbers. The United States Department of Agriculture, through the person in charge of leaf rust race identification (C. O. Johnston), has agreed to accept this responsibility. Where a local set of supplemental differentials is used, the number will be assigned by the worker who made up and published the set.

Thus, for example, a designation such as 9-NA59-1 would identify a culture recognized as race 9 on the eight standard differentials and as variant 1 on the North American supplemental differential set published in 1959. The designations 15-NA59-1 and 9-NA59-1 would indicate a difference in reaction on the standard differentials, but these cultures would have the same reaction on the supplemental differentials. On the other hand, 9-NA59-2 would differ from 9-NA59-1 only on the supplemental differentials. Such designations will be more complex than previous race numbers but will systematically indicate the phenotypic variation between different cultures as measured by the differentials used.

In the publication of critical research with leaf rust the author should use the complete designation including the race number based upon the eight standard differential varieties, but where the worker deems it expedient, he may publish designations based only on the supplemental differential set.

The system outlined will provide flexibility together with a uniform method of designating variation based upon supplemental differentials.

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ANTIBIOTICS AS SEED TREATMENTS FOR CONTROL OF GRAIN SMUTS[1]

Willard F. Crosier

Abstract

Acti-dione, three derivatives of Acti-dione, Agri-mycin and griseofulvin were compared with a volatile organic mercurial, Ceresan M, in the control of seed-borne smuts and seed decay of grains and grasses. None of the antibiotics effected satisfactory control of head smut of field bromegrass. Cycloheximide and the acetate, semicarbazone and oxime of cycloheximide reduced the incidence of oat smuts and nearly eliminated stinking smut of wheat. Agri-mycin and griseofulvin provided slight control. In laboratory germination tests the acetate and semicarbazone derivatives were phytotoxic to field bromegrass and Yorkwin wheat.

INTRODUCTION

Antimicrobial extracts from certain species of mold fungi, or rarely from soil, classified loosely as antibiotics, have been used as bactericides in diseases of animals for at least 15 years. The application of antibiotics to the control of bactéria in plants is more recent and only moderately successful. Seed treatment by antibiotics is still more recent and also less successful, especially if fungi are involved.

The first extract of antifungal nature, discovered in 1946 (7) and isolated in 1947 (4) as Acti-dione (cycloheximide) is now used as a biocide in the control of both bacterial and fungal diseases of plants. Ford et al. (1) in describing the agricultural uses of Acti-dione cited 135 publications. This antibiotic was mentioned as effective against diseases of alfalfa, apple, bean, broccoli, cabbage, cedar, cherry, corn, cucurbits, dewberry, grape, juniper, mint, mushroom, oak, orchid, pine, potato, rose, strawberry, turf grasses, and wheat.

Acti-dione and certain of its derivatives were reported by Hacker and Vaughn (2) as inducing resistance in wheat to stem rust without affecting the germinability of the seed crop. Vaughn et al. (6) soaked seeds of cantaloupe, cucumber, and spinach in solutions containing 100 ppm of Acti-dione for 30 minutes. The seeds germinated normally. Other seeds, especially wheat, were injured during 3-hour soaking periods in 20 ppm solutions. Henry et al. (3) found that barley and oat seed tolerated, while wheat seed suffered from, soaking in a 10 ppm solution. Dusts containing 0.5 to 1.0 percent of Acti-dione applied to wheat seed did not significantly reduce germination except with the highest dosage.

As a seed treatment for the control of grain smuts, Acti-dione was found by Henry et al. (3) to be partially effective but inferior to mercurial fungicides. In 1954, Machacek (5) reported that dusts containing Acti-dione reduced, but did not eliminate, smuts of barley, oats, and wheat. The materials did not effect an appreciable increase in field stands from treated flax and wheat seeds.

The seeds treated and distributed by Dr. J. E. Machacek of Winnipeg, Manitoba were received and tested at Geneva, New York. The results of these and more recent studies initiated by the writer are presented in this article.

MATERIALS AND METHODS

Seeds of Sanalta barley, Royal flax, Vanguard oats, and Thatcher wheat received from Winnipeg, Manitoba had been treated with dust formulations of 0.5 and 1.0 percent Acti-dione at a single dosage rate, 0.5 ounces of material per bushel of seed. About 6 weeks elapsed between treating and planting in quadruplicated rod rows at Geneva, New York.

Seedling stands were counted at the four-leaf stage of wheat and 1 month after sowing of the flax seed. Normal and smutted (*Ustilago nigra* Tapke and *U. nuda* (Jens.) Rostr.) heads of barley were counted from all plants in the four rod rows of each treatment and checks. Healthy and smutted (*Ustilago avenae* (Pers.) Rostr. and *U. kolleri* Wille) panicles of oats and healthy and smutted (*Tilletia foetida* (Wallr.) Liro) heads of wheat were also observed and recorded from four rods of rows.

[1] Approved by the Director for publication as Journal Paper No. 1158 on April 21, 1959.

In September 1956 seeds of field bromegrass, Genesee wheat, and Yorkwin wheat were treated with 1 percent active dusts of Acti-dione[2] and its acetate (A), semicarbazone (S), and oxime (O) derivatives. These materials as well as 33 percent Agri-mycin-100[3] and 5 percent griseofulvin[4] were applied at a dosage rate of 2 ounces of dust per bushel of seed.

Rod rows of each lot of antibiotic-treated seeds were planted in quadruplicate 10 days after treatment. The plantings also included untreated and mercurial (26 materials) treated seeds. The healthy and diseased seeds were counted during July of 1957.

One-pint lots of the seeds were stored in tightly-closed jars. Germination tests on paper towels and in contaminated soil were conducted after 7 and 11 months for the bromegrass and wheat seeds, respectively.

In April 1957 Anthony oats artificially inoculated with spores of Ustilago avenae and U. kolleri were treated with the six antibiotic preparations and Ceresan M. The rates of application were 1, 1 1/2, and 2 ounces per bushel of seed for all antibiotics and 1/4, 3/8, and 1/2 ounces for Ceresan M. The seed was planted either 8 days after treating in 1957 or 12 months later in May 1958. Naturally inoculated seed was treated and planted similarly.

RESULTS AND DISCUSSION

Dust applications of only 0.0025 ounces of actual Acti-dione per bushel of seed were ineffective in reducing seed decay of flax and seedling blight (Helminthosporium sativum Pam., King & Bakke) of wheat. As shown in Table 1 doubling the dosage was not beneficial. The reduction of smutted heads due to Acti-dione was recorded as negligible, partially effective and moderately effective for barley, oats, and wheat, respectively.

Table 1. Acti-dione in the control of smuts and seed rots of small grains.

Disease	Percent smut or stand of plants			
	0.5% Acti-dione	1.0% Acti-dione	Ceresan M	None
Barley smut	3	3	1	3
Oat smut	6	5	Tr	10
Wheat smut	4	3	Tr	27
Flax seed	17	15	33	20
Wheat seedling blight	66	69	76	65

Since the spores of Ustilago bullata Berk. are, in part, carried beneath the glumes of field bromegrass seeds, it is not surprising that the antibiotics did not materially reduce the incidence of head smut. As shown in Table 2, Acti-dione S was slightly more fungicidal than the other antibiotics but was very inferior to Ceresan M. In previous experiments only mercurials and formaldehyde have controlled this disease.

The surface borne spores of Tilletia tritici were almost completely killed by Acti-dione and its derivatives. As shown in Table 2, the derivatives were more fungicidal but, unfortunately, also more phytotoxic than Acti-dione to Yorkwin wheat and especially to field bromegrass. This is contrary to the findings of Hacker and Vaughn (2) who reported that Acti-dione alone was injurious to seedlings of Onas spring wheat.

The performance in soil at Geneva, New York indicated that the antibiotic preparations did not injure the seeds appreciably but neither did they afford protection from soil-resident organisms. The latter statement was supported by a parallel planting of seeds that received a supplementary treatment of captan immediately prior to planting. The addition of captan resulted in a slightly higher percentage of emergence in the Ceresan M-treated lots and in a significantly higher (10 to 27) percentage of seedlings in the antibiotic-treated lots. The average percents of emergence from Genesee and Yorkwin wheat, respectively, were: Acti-dione, 92 and 92; Acti-dione A, 90 and 91; Acti-dione S, 85 and 86; Acti-dione O, 90 and 94; Agri-mycin, 95 and 94; griseofulvin, 91 and 92; Ceresan M, 89 and 91; and untreated, 89 and 90.

Since the fungi of oat smuts are carried beneath the palea and lemma enclosing the seed, they are not easily reached by solid materials. Chemicals that volatilize readily such as for-

[2] All Acti-dione materials supplied by the Upjohn Company, Kalamazoo, Michigan.

[3] Supplied by Chas. Pfizer and Company, Inc., Brooklyn, New York. The diluted material contained 5 percent streptomycin and 0.5 percent Terramycin.

[4] Supplied by Merck and Company, Inc., Rahway, New Jersey.

Table 2. Control of grain smuts and of seed decay by, and phytotoxicity of, antibiotics when dusted on bromegrass and wheat seeds.

Antibiotic present in dust	Percent of smutted heads			Percent germination on paper towels			Percent emergence from contaminated soil		
	Brome	Genesee	Yorkwin	Brome	Genesee	Yorkwin	Brome	Genesee	Yorkwin
Acti-dione	57	1	2	92	95	92	61	65	69
Acti-dione A	60	1	1	7	93	59	48	68	73
Acti-dione S	53	1	1	69	90	49	55	72	72
Acti-dione O	55	Tr	1	84	93	82	55	75	77
Agri-mycin	81	5	47	93	93	95	56	68	84
Griseofulvin	66	7	33	95	95	94	51	72	79
Ceresan M	3	0	Tr	82	94	91	42	81	86
Check	76	16	63	93	92	95	45	67	78

Table 3. Control of oat smut by seed treatment with antibiotics and another fungicidal dust.

Material applied to seeds	Percentage of smutted panicles						
	Seed as harvested		Seed with spores added				
	Sown in 1957		Sown in 1957			Sown in 1958	
	Full[a]	Half	Full[a]	3/4ths	Half	Full[a]	3/4ths
Acti-dione	Tr	1	5	7	6	3	2
Acti-dione A	Tr	Tr	2	2	2	0	Tr
Acti-dione S	Tr	Tr	3	7	6	Tr	2
Acti-dione O	Tr	1	4	4	8	1	4
Agri-mycin	1	1	5	11	13	9	11
Griseofulvin	1	1	5	7	8	13	18
Ceresan M	0	0	1	Tr	Tr	0	Tr
Check	3	3	14	14	14	17	17

[a] Full refers to the normal dosage rate of 2 ounces and 0.5 ounce per bushel of seed for the antibiotics and Ceresan M, respectively.

maldehyde, and certain organic mercurials are effective as oat seed treatments.

The data in Table 3 indicate that Acti-dione acetate is more effective than the other antibiotics in the control of oat smuts. The mode of action is uncertain but control may be accomplished through volatility. However, the systemic action of Acti-dione derivatives is recognized in control of certain foliage diseases and may be operative here. When the percentages of smutted panicles recorded for 1958 are compared with those observed in 1957, it is apparent that regardless of the mode involved, storage enhanced the fungicidal action of only the Acti-dione preparations.

The phytoxicity of the antibiotics was not studied with oat seeds. It was negligible, however, since excellent stands were obtained in all rows in the 1958 plantings.

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NEW YORK STATE AGRICULTURAL EXPERIMENT STATION, CORNELL UNIVERSITY, GENEVA, NEW YORK

SUSCEPTIBILITY OF ALFALFA VARIETIES TO PHYSODERMA ALFALFAE[1]

Charles M. Leach[2] and John R. Hardison[3]

Summary

Field plots of 24 varieties of Medicago sativa and a single seed lot of M. falcata were artificially inoculated with the crown wart fungus, Physoderma alfalfae. All the varieties were susceptible to P. alfalfae; however, the degree of susceptibility was significantly different between the varieties. The most tolerant varieties were Talent, Socheville, Du Puits, Sevelra, Vernal, Williamsburg, Cossack, Atlantic and Grimm, while the most susceptible group included Nomad, Narragansett, Ladak, Rhizoma, Ranger, Synthetic A-169, Buffalo, Lahontan, Uruguay (clone 10) and Indian.

Crown wart of alfalfa (2), caused by the fungus Physoderma alfalfae (Lagh.) Karling, has been a major problem for many years on low lying, wet land in western and southwestern Oregon (3). The disease is quite restricted and economically is of minor importance to the State's total alfalfa production.

FIGURE 1. Alfalfa plants severely infected with crown wart.

1 Technical Paper No. 1208, Oregon Agricultural Experiment Station.
2 Assistant Plant Pathologist, Department of Botany and Plant Pathology, Oregon Agricultural Experiment Station.
3 Pathologist, Forage and Range Branch, Agricultural Research Service, United States Department of Agriculture.

Table 1. Susceptibility of alfalfa varieties to the crown wart fungus *Physoderma alfalfae.*

Variety[a]	Disease rating[b]	Significance[c] 5%	1%
Talent	.53		
Socheville	.60		
Du Puits	.73		
Sevelra	1.17		
Vernal	1.23		
Williamsburg	1.23		
Cossack	1.27		
Atlantic	1.37		
Grimm	1.37		
Pilca Butta	1.53		
Orestan	1.60		
Meeker Baltic	1.63		
Nomad	1.67		
Narragansett	1.77		
Ladak	1.80		
Rhizoma	1.87		
Ranger	2.00		
Synthetic A-169	2.13		
Buffalo	2.13		
Lahontan	2.33		
Uruguay (clone 10)	2.37		
Indian	2.53		

[a] African and *M. falcata* are excluded from this table because of insufficient plants for adequate sampling. Both varieties were susceptible to *Physoderma*.
[b] Disease rating: 0 = no warts, 1 = slight warting, 2 = moderate warting, 3 = severe warting, and 4 = plants killed by crown wart.
[c] Varieties bracketed by the same line are not significantly different.

Alfalfa varietal trials conducted in western Oregon revealed that some varieties sown on land infested with the crown wart fungus were less susceptible than others. In 1954 a field experiment was specifically designed to determine the relative susceptibility of 24 varieties of Medicago sativa and a single seed lot of M. falcata to the crown wart fungus. The alfalfa varieties were established[4] during August on a sandy loam soil, in single 20-foot rows sown 12 inches apart and replicated three times in a randomized block design. The varieties sown were African, Atlantic, Buffalo, Caliverde, Cossack, Du Puits, Grimm, Indian, Ladak, Lahontan, Meeker Baltic, Narragansett, Nomad, Orestan, Pilca Butta, Ranger, Rhizoma, Sevelra, Socheville, Talent, Uruguay (clone 10), Vernal, Williamsburg, Synthetic A-169 and M. falcata. The following autumn the varieties were inoculated by dusting spores over the plants in the seedling rows. Spore inoculum was prepared by grinding galls from diseased plants in a household meat grinder.

The varieties were scored for disease damage in July 1957, three years after establishment of the plots. Ten plants were dug at random from each of the replicated rows and rated for severity of infection (Figure 1). Stand and yield data were not used, as they were affected by winter injury and damage from root rot organisms.

RESULTS AND DISCUSSION

None of the varieties tested were completely resistant to Physoderma alfalfae (Table 1). However, the data, when analyzed by the "Multiple Range Test" of Duncan (1), revealed significant differences between the varieties at both the 5 and 1 percent levels. The most tolerant group of varieties included Talent, Socheville, Du Puits, Sevelra, Vernal, Williamsburg, Cossack, Atlantic and Grimm; while the most susceptible group included Nomad, Narragansett, Ladak, Rhizoma, Ranger, Synthetic A-169, Buffalo, Lahontan, Uruguay and Indian. There were conspicuous differences in vegetative growth between the most susceptible and tolerant varieties.

These trials have confirmed the hypothesis that different alfalfa varieties vary in susceptibility to P. alfalfae. Several of the varieties commonly grown in western and southwestern Oregon are varieties which were the most tolerant in this investigation. These varieties have been recommended because they have yielded and persisted better than other varieties.

Although the resistance of the more tolerant varieties can probably be further increased through breeding and selection, the limited extent of crown wart in Oregon hardly justifies this course of action. At present, therefore, it is recommended that alfalfa growers on infested land grow only those varieties shown to be most tolerant in this study.

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

4 Appreciated is the advice and assistance of Mr. H. H. Rampton, Agronomist, United States Department of Agriculture, who established the varietal plots.

A SURVEY OF ROOT DETERIORATION OF MEDICAGO SATIVA IN OREGON[1]

Charles M. Leach[2]

Summary

A survey was made of the occurrence and severity of root deterioration of Medicago sativa in Oregon. Fifty-nine percent of the roots sampled showed vascular discoloration, 39 percent showed superficial discoloration of the cortical tissues and 64 percent were injured by insects, presumed to be Sitona hispidula. The incidence and severity of root deterioration varied from region to region, and increased with the age of the planting. Three species of fungi were consistently isolated from discolored root-steles; these were Phoma herbarum var. medicaginis (42 percent), Fusarium solani (29 percent) and F. oxysporum (16 percent).

Alfalfa (Medicago sativa L.) is grown extensively in Oregon for hay and to a much lesser extent for seed. During the spring of 1957 a survey was made to determine the principal diseases of this crop. As information on the nature and extent of root diseases of alfalfa within the State is negligible, main emphasis of this survey was restricted to these disorders.

It was impractical to survey all alfalfa-growing regions so sampling was restricted mainly to Benton, Jackson, Umatilla and the central Oregon counties (Crook, Deschutes and Jefferson). Less intensive surveying was conducted in a few other widely scattered areas. The regions were selected because of their diversity of climates, topography and cropping practices. The varieties of alfalfa surveyed included Talent, Ranger, Ladak, Vernal, Lahontan, Du Puits, Narragansett, Sevelra and several unidentified varieties. The ages of alfalfa stands sampled ranged from 1 to 4 or more years. When the age was not known, an estimate was made.

PROCEDURE

Ten to 30 plants were randomly dug from each field and, where possible, at least 1 foot of tap root was obtained. In non-irrigated fields, because of difficulty of digging, shorter lengths of roots were sometimes obtained. Sampling was restricted to tap roots in all but the youngest stands. Roots were placed in polyethylene bags and later examined macroscopically in the laboratory for insect injury, vascular and cortical discoloration or decay. The extent of vascular discoloration was determined by splitting the roots longitudinally at several locations along their lengths. The severity of the three different disorders, that is, insect injury, vascular discoloration, and cortical discoloration or decay, were rated separately as follows: 0 = healthy; 1 = slight decay, discoloration or injury; 2 = moderate decay, discoloration or injury; 3 = severe decay, discoloration or injury.

After visual examination, isolations were made from those roots showing vascular discoloration. No isolations were made from roots showing cortical discoloration, for this condition, though extensive, was superficial. Neither were isolations made from cortical decays, for these were rare and when present were restricted to the crowns and typical of winter injury.

Roots with vascular discoloration were scrubbed under running water and pieces of the tap roots approximately 1 inch in length were excised. The cortical tissues of the pieces were removed and the steles placed in a 1:4 mixture of "clorox" (5.25% sodium hypochlorite) and water for 5 to 20 minutes depending upon the size of the roots. They were then thoroughly washed in sterile water, placed in a 150 ppm solution of streptomycin nitrate, and a vacuum was applied with a high vacuum pump for 5 minutes to infiltrate the tissues. The roots were infiltrated with streptomycin because of the abundance of bacteria which tended to inhibit fungal growth from within the stele. Finally the roots were cut into five or more pieces, placed on potato-dextrose agar having an unadjusted pH of 5.3-5.8 and incubated for 10 days at room temperature in diffused daylight.

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[1]Technical Paper No. 1218, Oregon Agricultural Experiment Station.
[2]Assistant Plant Pathologist, Department of Botany and Plant Pathology, Oregon Agricultural Experiment Station.

RESULTS

The tap roots of 1050 alfalfa plants from 59 fields were examined macroscopically for vascular discoloration, cortical discoloration or decay, and insect injury. Table 1 is a summary of the type of deterioration observed and Table 2 shows the fungi isolated from the roots. Tables 3 and 4 are the same data showing the relationship of plant age to type and severity of root deterioration, and species of fungi isolated.

Table 1. Incidence and severity of root deterioration in alfalfa.

Location	Number of fields sampled	Root symptoms[a]: Vascular discoloration: Disease index	Vascular discoloration: Percent diseased	Cortical discoloration or decay[b]: Disease index	Cortical discoloration or decay: Percent diseased	Insect injury: Injury index	Insect injury: Percent injured	Number of plants examined
Benton County	10	0.38	28	0.07	38	1.82	94	260
Central Oregon	13	1.23	72	0.49	20	0.59	30	310
Jackson County	10	1.66	75	1.46	53	1.28	54	94
Umatilla County	9	0.84	45	1.00	49	1.85	83	124
Miscellaneous	17	1.57	73	0.99	37	1.43	60	262
Total	59							1050
Mean		1.17	59	0.66	39	1.31	64	

[a]Insect injury and disease indices: 0 = healthy; 1 = slight decay, discoloration or injury; 2 = moderate decay, discoloration or injury; 3 = severe decay, discoloration or injury.
[b]Mainly a superficial cortical discoloration.

Table 2. Species of fungi isolated from alfalfa roots showing vascular discoloration[a].

Location	Fungi isolated[b] (in percent): Fusarium oxysporum	Fusarium solani	Fusarium spp.	Phoma herbarum var. medicaginis	Unidentified	Number of plants isolated from	Number of fungi isolated
Benton County	10	50	10	30	0	42	20
Central Oregon	6	25	6	52	11	107	134
Jackson County	25	28	0	37	10	59	79
Umatilla County	33	29	0	29	8	50	24
Miscellaneous	7	15	6	60	12	119	83
Mean	16	29	4	42	8		

[a]Only known pathogenic fungi are included.
[b]Percent of the total root isolations for each location.

Vascular Discoloration

Discoloration of vascular tissues ranging from slight to severe was evident in 59 percent of the plants examined (Table 1). In some fields practically 100 percent of the plants were affected. This type of root deterioration was least severe in Benton County where 28 percent of the plants were affected (disease index 0.38) and most severe in Jackson County where 75 percent of the plants were affected (disease index 1.66). There was a noticeable increase in vascular discoloration of the roots with increase in age of the plants (Table 3). Discoloration in roots of 1-year-old plants was 32 percent (disease index 0.42), contrasting with 83 percent (disease index 1.78) for plants that were 4 years or more old.

Table 3. Age of alfalfa plants and severity of root deterioration[a].

Age of plants (years)	Number of fields sampled	Root Symptoms[b]						Number of plants examined
		Vascular discoloration		Cortical discoloration or decay		Insect injury		
		Disease index	Percent diseased	Disease index	Percent diseased	Injury index	Percent injured	
1	16	0.42	32	0.02	1	1.04	53	443
2	9	1.16	62	1.22	47	1.06	51	121
3	8	1.67	83	1.24	50	1.72	77	137
4+	17	1.78	83	1.34	53	1.50	68	246
Total	50							947

[a]Data from nine fields excluded because age of stands unknown.
[b]Insect injury and disease indices same as in Table 1.

Table 4. Age of alfalfa plants and fungi isolated from discolored root steles[a].

Age of plants (years)	Fungi isolated[b] (in percent)					Number of plants isolated from	Number of fungi isolated
	Fusarium oxysporum	Fusarium solani	Fusarium spp.	Phoma herbarum var. medicaginis	Unidentified		
1	7	30	6	55	3	72	71
2	3	10	10	66	10	50	29
3	14	20	5	47	14	77	74
4+	21	33	3	40	4	115	107

[a]Data from nine fields excluded because age of stands unknown.
[b]Percent of total isolations for each age group.

Cortical Discoloration or Decay

Discoloration of the cortical tissues was present in 39 percent of the plants examined (Table 1). Roots from Benton County and central Oregon showed the least amount of discoloration or decay with 38 percent (disease index 0.07) and 20 percent (disease index 0.49) affected, respectively. In contrast 53 percent (disease index 1.46) of the roots from Jackson County were affected. Where this type of deterioration was present, it appeared as a rusty-brown discoloration of the root surfaces which, though often extensive, was quite superficial in nature. The incidence and severity of cortical discoloration or decay was much lower for 1-year-old plants than for older plants.

Insect Injury

The type of insect injury observed consistently appeared as cavities and grooves in the cortical tissues of the tap root, and was typical of that caused by the clover-root curculio, Sitona hispidula (Fabr.). Insect injury was present in 64 percent (injury index 1.31) of the roots sampled. Roots from central Oregon were the least injured, with 30 percent (injury index 0.59) affected. In Umatilla and Benton counties insect injury was more widespread and 83 percent (injury index 1.85) and 94 percent (injury index 1.82) of the roots were affected, respectively. The incidence and severity of insect injury generally was somewhat less in 1- and 2-year-old stands than in older plantings.

Fungi Associated with Vascular Discoloration

Three species of fungi were consistently isolated from discolored vascular tissue (Table 2); these were *Phoma herbarum* var. *medicaginis* West. ex Rab., *Fusarium solani* sensu Snyder & Hansen, and *F. oxysporum* sensu Snyder & Hansen. Forty two percent of the fungi isolated were *P. herbarum* var. *medicaginis*, 29 percent *F. solani* and 16 percent *F. oxysporum*. All three fungi occurred in discolored steles of both young and old plants in approximately the same proportions (Table 4).

DISCUSSION OF RESULTS

Insect injury presumed to be caused by *Sitona hispidula* and vascular discoloration were the most prevalent symptoms of root deterioration in Oregon plantings of alfalfa. These two conditions, particularly in older stands, appear to be associated with the declining yields of alfalfa plantings. Although cortical discoloration was also widespread, it was practically always of a superficial nature with apparently no penetration into the cortical tissue. It is doubtful that this discoloration causes any retardation of plant growth. Where true cortical decays were encountered they were restricted to the portion of the root just below the crown and were typical of winter injury rather than injury of pathogenic origin.

The fungi isolated from vascular tissues in this survey have all been reported to be active pathogens of alfalfa (1, 2, 3, 4, 5, 6, 7). However, it cannot be assumed that these species were the sole cause of vascular discoloration, for both the bacterial wilt organism, *Corynebacterium insidiosum* (Mc Cull.) H. L. Jens., and several viruses cause discoloration of vascular tissue. The high incidence of *Phoma herbarum* var. *medicaginis* within the vascular tissue of roots is surprising for the fungus is more usually considered to be a pathogen associated with the above-ground parts of the alfalfa plant, although it has been isolated from roots (2). The pathological significance of the presence of this fungus in the vascular tissues is a matter of speculation.

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OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

GLIOCLADIUM, A CAUSAL AGENT IN THE BEAN ROOT ROT COMPLEX IN IDAHO[1]

D. M. Huber[2] and A. M. Finley[3]

Chatterjee (1) demonstrated the principal pathogenic agent involved in bean root rot in Idaho to be Fusarium solani f. phaseoli (Burk.) Snyder & Hansen. Rhizoctonia solani Kuehn was isolated frequently from diseased roots but pathogenicity studies indicated that most of the isolates were either weakly pathogenic or non-pathogenic. Research on bean root rot has continued in Idaho and in 1957 isolations from diseased roots of plants of the Red Mexican variety yielded a high percentage of Gliocladium roseum (Link) Thom. Although G. roseum was often associated with Fusarium spp., approximately 34 percent of the isolates were pure cultures. G. roseum was also isolated frequently from the soil of experimental field plots by the use of a sampling tube technique similar to that reported by Mueller and Durrell (2).

Species of Gliocladium are known to be common inhabitants of the soil but they have rarely been reported to cause diseases of higher plants. Chatterjee (1) was unable to demonstrate pathogenicity by the isolates of Gliocladium which she obtained, and considered them to be contaminants or secondary invaders. G. aureum sp. nov. was reported by Rader (3) to cause a storage rot of carrots. No other reference to Gliocladium spp. as causal agents of disease could be found in the literature.

The frequency with which Gliocladium roseum was isolated in pure culture from diseased beans grown in the field prompted further study of its role in the bean root-rot disease in Idaho.

MATERIALS AND METHODS

Isolates of Gliocladium roseum obtained from infected bean roots were sub-cultured by making four to five serial transfers to insure generic purity. After these sub-cultures had been carefully examined microscopically and found to be free from mixtures of other organisms, single spores were isolated and cultured. Cultures of G. roseum thus obtained were then used in pathogenicity studies. The test host in these experiments was Red Mexican bean of the UI-34 strain. Screening tests were conducted in the laboratory using plants that were grown in nutrient solution according to the procedure described by Chatterjee (1). These plants were inoculated when they were approximately 6 days old by placing mycelial mats of the fungus at the base in the hypocotyl. The plants were grown in the laboratory at room temperature and observed daily for the appearance of necrotic lesions. When lesions developed, the diseased portions of the roots and hypocotyl were surface-sterilized for 10 minutes in a 30 percent Clorox solution and plated on corn meal agar. The agar plates were incubated at 20° C. The reisolates thus obtained were compared with the originals.

Subsequent pathogenicity tests using a virulent isolate selected from the previous test were conducted in the greenhouse. A randomized block design was used for these tests with five replicates of five pots each and five plants per pot. Duplicate tests were conducted using steamed sand and steamed field soil respectively. The steamed substrate in each case was artificially infested with inoculum of a pure culture of Gliocladium roseum grown on steamed oats. After the substrates were infested with the organism, bean seed was surface-sterilized and planted. Five weeks after emergence, the plants were removed from the pots and roots were examined for evidence of rot. The diseased roots were then taken to the laboratory, sectioned with a freezing microtome, mounted in a lactophenol-cotton blue medium and examined under the microscope. Observations were made to determine the presence of G. roseum, the mode of penetration, and the distribution of the mycelium within the plant tissues. Isolations were also made from these roots to establish proof of the identity of the organisms present.

RESULTS

Sixty-four plants were selected at random from root-rot field study plots and isolations were made to determine the identity of pathogens involved. Twenty-nine of the 64 isolates

[1]Published with the approval of the Director of the Idaho Agricultural Experiment Station as Research Paper No. 467.

[2]Formerly Research Assistant Plant Pathologist, Idaho Agricultural Experiment Station.
[3]Plant Pathologist, Idaho Agricultural Experiment Station.

were pure cultures of Fusarium solani f. phaseoli, 21 were pure cultures of Gliocladium roseum and 14 yielded mixed cultures of F. solani f. phaseoli and G. roseum.

Six of the 21 isolates of Gliocladium roseum were tested for pathogenicity in the laboratory and five of these produced lesions on the hypocotyls of the test plants. Evidence of infection was apparent after a 48-hour exposure to the inoculum. Within 5 days after inoculation, all of the plants except those exposed to the one non-pathogenic isolate exhibited symptoms of disease. In the greenhouse tests, initial symptoms of disease were observed 3 weeks after the plants emerged. These symptoms consisted of small linear lesions on the hypocotyl and primary roots. The lesions enlarged, and coalesced to encompass the hypocotyls and roots and then progressed up the hypocotyl to the ground line (Fig. 1). The lesions are reddish-brown in color at the outset and become a dark-brown with age. Lateral or secondary roots were usually found to be severely infected and were frequently destroyed soon after they formed. Examination of cross sections of the hypocotyl revealed the mycelium to be generally distributed throughout the cortical tissue both within and between the cells. Hyphae were observed in the plant hairs on the epidermis and limited penetration was observed in the vascular elements. The vascular elements were discolored and appeared to be plugged in a manner similar to that which is usually observed in the case of vascular wilt diseases. The root rot caused by this organism differs in this respect from that caused by Fusarium solani f. phaseoli. Beans that were grown in a sand substrate in the greenhouse developed more severe symptoms of root rot than did those grown in a soil substrate. The disease seemed to progress more rapidly also in the sand cultures.

FIGURE 1. Symptoms of Gliocladium root rot on Red Mexican bean, 5 weeks after planting.

The isolates of Gliocladium roseum used in these tests produced a yellow pigment when they were cultured on corn meal agar in the absence of light. When they were cultured on corn meal agar and exposed to daylight, the pigment produced was pink. Cultures incubated in the dark for a period of 36 to 72 hours produced a yellow pigment, but when these cultures were then exposed to daylight for a period of 24 hours or more, the pigment changed to a pinkish-orange. When the isolates were cultured in daylight for a period of 36 to 72 hours, the pigment was pink and these cultures remained pink even though they were subsequently transferred to a dark incubation chamber. Isolates cultured on PDA and Czapek's agar in the daylight had an orange pigmentation. When they were incubated in the dark they produced a light-pink pigment on Czapek's agar and no pigmentation on PDA. The cardinal temperatures for these isolates were found to be -- minimum 10° C, optimum 20° to 28°, and maximum 32°.

These results indicate quite conclusively that strains of Gliocladium roseum, pathogenic to beans, are present in the soils of Idaho. The root-rot symptoms caused by G. roseum closely approximate those caused by Fusarium solani f. phaseoli. Observations made during the course of these investigations led the authors to believe that F. solani f. phaseoli is a more virulent pathogen than G. roseum. Thus, if the inoculum potential for both organisms is equal,

F. solani f. phaseoli will, under most conditions, invade the roots and hypocotyl of bean plants more rapidly than will G. roseum; therefore, it will be the most destructive organism. On the other hand, if the potential is unequal, root rot symptoms may be produced by whichever organism is the most prevalent.

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IDAHO AGRICULTURAL EXPERIMENT STATION, MOSCOW

DROUGHT SPOT OF LETTUCE COTYLEDONS[1]

A. M. Finley[2]

Seed analysts commonly report necrotic spot on the cotyledons of lettuce seedlings and generally believe them to be indicative of inferior quality. Those seedlings that exhibit large spots on the cotyledons are usually reported as abnormal and are discounted on the germination record. The existence of an appreciable number of spotted plants from any given lot of seed can result in financial loss to the producer, because crops are sold on the basis of the germination record.

Cotyledonary spots of variable size, shape, and color have been reported to occur on lettuce. The red spotting that Dempsey and Harrington[3] reported on seedlings in California has not been observed in Idaho, but black and brown spots are commonly found. The development of these spots has been attributed to many causes, including: insects, pathogenic agents, drought, heat and poor storage conditions.

This malady is apparently universal in occurrence but the symptoms and mode of expression are variable. In Idaho spotting is frequently observed in germinated seedlings from freshly harvested seeds but damage may be aggravated by long periods of unfavorable storage conditions.

MATERIALS AND METHODS

This study was initiated by a thorough analysis of the lettuce seed germination records of the Idaho State Seed Laboratory for the 3 year period 1951 through 1953. The incidence of cotyledonary spotting was correlated with varieties, varietal strains and season.

Laboratory Studies

Seed lots that were reported to contain a high percentage of affected cotyledons were traced to their source and samples of those lots still available were obtained for laboratory study. A second germination analysis was run in our laboratory according to the official rules of the Association of Seed Analysts. Seedlings that exhibited necrotic spots on the cotyledons were removed from the germinator and isolations were attempted to determine the presence of pathogenic organisms. Affected cotyledons were surface sterilized with a solution of $HgCl_2$ (1:1000 aqueous) and plated on corn meal agar. Other discolored cotyledons were embedded in paraffin, sectioned, and examined for signs of microorganisms or cellular inclusions which might indicate the presence of virus particles.

Field Studies

The lettuce aphid *Macrosiphum barri* Essig commonly infests lettuce florets in Idaho; therefore, controlled experiments were conducted to determine whether that insect might be a causative factor in this disease. The experiments were conducted with the cooperation of Mr. A. J. Walz[4] on the Parma Branch of the Idaho Agricultural Experiment Station. A paired plot design was used with six varieties and five replications of each. Parathion was applied to one of each pair of plots as needed to keep the aphid population to a minimum. Aphids were permitted to multiply freely in the unsprayed plots. A census of the population was taken periodically by collecting floral tips and counting the number of aphids on each. Seed samples were collected from these plots at harvest time, germinated in the laboratory, and examined for seedling damage.

Greenhouse Studies

Weather as a factor in crop production is subject to considerable seasonal variation; therefore, its more important elements, temperature and moisture, were studied under con-

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[1] Published with the approval of the Director of the Idaho Agricultural Experiment Station as Research Paper No. 466.
[2] Plant Pathologist, Department of Plant Pathology, University of Idaho.
[3] Dempsey, W. H., and J. F. Harrington. 1951. Red cotyledon of lettuce. California Agr. 5(7): 4.
[4] Formerly Assistant Entomologist, University of Idaho.

trolled conditions in the greenhouse to determine their role in the development of cotyledonary necrosis. Lettuce plants of the Black Seeded Simpson variety were grown under a controlled temperature of 80° ± 2° F and optimum soil moisture levels until seed stalks were formed and flower buds became well defined. At this stage of development, the plants were divided into four lots of five plants each. Lots 1 and 2 were left on the greenhouse bench to develop seed under the influence of maximum daily temperatures within the range of 80° to 90° F. Lots 3 and 4 were placed in a temperature chamber where the maximum daily temperature ranged from 100° to 110°. Lots 1 and 3 were irrigated as needed to keep the soil moisture level just above the wilting percentage. The soil moisture level was maintained at near the maximum capacity for lots 2 and 4. When the seed had matured on these plants, it was harvested and analysed in the laboratory.

RESULTS

Careful study and analysis of the data obtained from the State Seed Laboratory indicated that there are no significant differences between varieties or varietal strains in their susceptibility to spotting. These analyses suggested a seasonal influence which is illustrated by the data presented in Table 1.

Table 1. Cotyledonary spotting of lettuce seed crops as determined by Idaho State Seed Laboratory analysis.

Classes (percent spotting)	Number of crops examined 1951	1952	1953
0.0	24	4	4
0.1 - 2.4	1	40	31
2.5 - 4.9	0	27	7
5.0 - 7.4	1	12	1
7.5 - 9.9	1	10	0
10.0 - 15.0	1	6	0
15.1	0	5	0
Maximum (percent)	10.00	33.75	7.25
Total number of crops	28	104	43

The rather obvious effect of season upon the incidence of spotting narrowed the field of investigation to three of the more probable variables, that is, weather, diseases, and insects. Weather data was obtained from the local U. S. Weather Reporting Station for the years 1951, 1952, and 1953 and analyzed. Temperature fluctuated within normal limits during all 3 years until July 22. In 1953 the temperatures continued to fluctuate within normal limits throughout the growing season. In 1951 and 1952 temperatures rose sharply on July 23 and 24 and continued above normal levels for a period of 1 week in 1951 and 3 weeks in 1952. Since July 22 to August 10 is the period of most rapid seed development, it is conceivable that abnormally high temperatures may have caused the excessive cotyledonary spotting observed in the 1952 crop. This hypothesis formed the basis for subsequent greenhouse investigations.

Two of the five seed lots tested in 1952 that contained a high percentage of affected plants were still being held in the owners' warehouses; therefore, samples were obtained for further analysis. The results of these analyses agreed rather closely with results reported by the State Seed Laboratory. There had been no apparent increase in spotting during the storage season. Seedlings exhibiting spotting were removed from the incubator and examined for the presence of pathogenic agents. Numerous attempts were made to isolate pathogenic organisms from the tissues contiguous with the necrotic areas but the results were all negative. Histological examination of affected cotyledons revealed no evidence of the presence of pathogens. It was concluded, therefore, that this disease was due to non-infectious causes.

Insect populations vary from season to season; therefore, field experiments were carried out to determine their role in this malady. These experiments were conducted for a period of 2 years, during which time temperatures fluctuated within normal limits for the area and the aphid population remained stable. Results of these experiments are presented in summary form in Table 2.

Table 2. Effect of Macrosiphum barri on cotyledonary spotting.

Variety	Percent: Germination	Abnormal[a]	Spotted	Dead	Aphid count[b] (Number)
New York 12	96	0	4	4	31
	97	1	2	2	0
Oak Leaf	93	0	2	7	76
	97	0	0	3	0
Big Boston	88	5	3	7	36
	97	1	7	2	0
B. S. Simpson	94	0	3	6	65
	96	1	5	3	0
Iceberg	97	1	0	2	66
	97	1	1	2	0
Grand Rapids	97	2	1	1	38
	98	0	1	2	0

[a]Abnormal seedlings were malformed and of low vigor.
[b]Average count per 2-inch floral tip.

The percentage of spotted cotyledons in these seed lots was quite low and there were no significant differences between treatments. Therefore, it was concluded that the aphid Macrosiphum barri was not responsible for the development of necrotic spots.

Greenhouse experiments were conducted to determine effect of temperature and soil moisture level upon the incidence of spotting. Results are summarized in Table 3.

Table 3. Effect of temperature and soil moisture level on cotyledonary spotting.

Soil condition	Maximum daily temperature	Percent: Germination	Abnormal	Spotted	Dead
Dry	100-110	47	30	52	14
Wet	100-110	96	1	6	3
Dry	80-90	99	0	1	0
Wet	80-90	0	0	1	0

These data indicate that high atmospheric temperatures, when combined with a soil moisture deficit during the formative stage of embryo development, will cause necrosis of the cotyledons and even death of the embryo in extreme cases. Histological examination of the necrotic tissues demonstrated that necrosis first occurred in the parenchyma cells adjacent to the conductive vessels. Cross sections of the necrotic cotyledons often revealed a band of necrotic cells around the vascular bundles (Fig. 1). In more severe cases cells of the mesophyll and palisade parenchyma were affected (Fig. 2). Large necrotic areas were often observed near the apex of the cotyledon and near the base or at the cotyledonary attachment. Apparently the reason for this is that the three veins of the cotyledons unite at the apex and lie adjacent to each other at this base.

CONCLUSION

It was concluded from the results of these studies that the cotyledonary spotting that develops on lettuce seed produced in Idaho is a manifestation of physiologic drought. The tissues of developing lettuce embryos are apt to become desiccated as a result of moisture stress, if maximum daily temperatures in excess of 90° F occur over an extended period of time and available soil moisture is low. Those cells and tissues immediately adjacent to the vascular elements are the first to succumb to this stress. If the stress continues over a long period of time, it is transmitted to more distant cells and in extreme cases the entire embryo may be killed. Lettuce seed fields are not customarily irrigated during this part of the season because of the danger of losses due to Sclerotinia sclerotiorum; therefore, the soil moisture level is usually low. The exact soil moisture content and, consequently, the magnitude

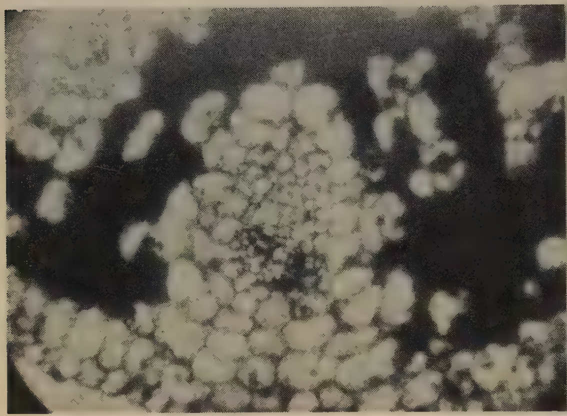

FIGURE 1. Band of necrotic tissue surrounding vascular bundle. (note necrotic cells within the bundle)

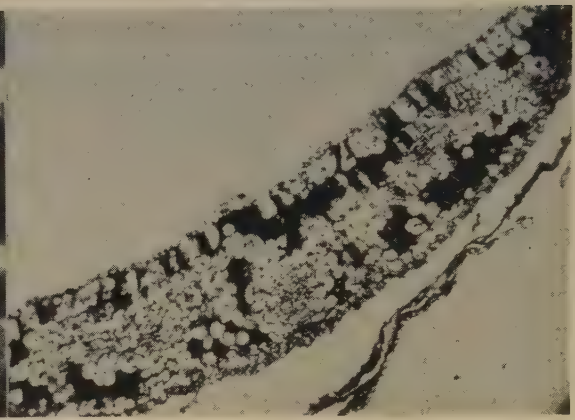

FIGURE 2. Cross section of cotyledon showing the distribution of necrotic cells.

of the moisture stress within the plants will vary with soil type and management. This probably accounts for the variability in incidence of necrotic spotting between crops.

IDAHO AGRICULTURAL EXPERIMENT STATION, MOSCOW

PENTACHLORONITROBENZENE AND UREA-FORMALDEHYDE FOR POTATO SCAB CONTROL IN MICHIGAN[1]

H. S. Potter, W. J. Hooker, W. Cargo, and G. T. Stachwick

Pentachloronitrobenzene (PCNB) and urea-formaldehyde were used in various Michigan soil types as broadcast and in-row soil treatments for control of potato scab Streptomyces scabies (Thaxt.) Waksman and Henrici. These results are in general agreement with previous reports of urea-formaldehyde (1) and of PCNB as scab control treatments (2, 3, 4, 6, 7, 8). This paper presents additional data on the effectiveness of certain soil treatments for the control of scab on potatoes. The studies described represent a phase in a continuing investigation which it is hoped will lead to an effective control program for this disease in Michigan.

All locations had a history of severe scab. The susceptible Chippewa variety was used throughout in plots of 0.01 A. Scab incidence was determined after a method previously described (5).

Uniform trials with PCNB in 1955 and 1956 were made with two 18-inch sets of disks mounted in tandem between the tractor wheels. PCNB was dispensed from a hopper in front of the disks in a band 12 to 14 inches wide and mixed with the soil in a single operation. A two-row planter was drawn behind the tractor and planting was completed at the same time. During both seasons potatoes grew very poorly in the various experimental trials because of irregular and deficient moisture supply. Mixing of PCNB with the soil in a single application was often not complete and control was poor. In general, this method of application was not practical because PCNB was not dispersed throughout the soil in which tubers were formed.

In a very sandy soil at Leelanau County a reasonably satisfactory degree of scab control was obtained with applications of 45 pounds of 100 percent active PCNB per acre (Table 1).

In Missaukee County application was made by introducing a rather low rate of PCNB with the above equipment and repeating the operation until a sufficiently heavy rate had been applied (Table 1). Under these circumstances the mixing was satisfactory and relatively good scab control was achieved.

In 1958, PCNB[2] was applied as a continuous broadcast treatment with a Howry-Berg All-Chem applicator (Fig. 1) at the rate of 50 pounds of active chemical per acre. This machine was mounted on the tool bar of a tractor equipped with a three-point hitch. The chemical was distributed in adjacent bands, 18 inches wide, over the entire plot surface then mixed, by means of a series of toothed wheels, into the soil to a depth of 4 to 6 inches.

Urea-formaldehyde (UF-85)[3] is a viscous liquid, which was diluted with water in a ratio of 1 to 4. It was applied to the soil with a sprinkling can at the rate of 150 gallons per acre. The surface of treated soil was not disturbed until planting the following day. There was one exception -- in Houghton County, soil was treated in an 18-inch band over the rows to be planted and the areas between the rows were left untreated. Potato seed was planted in the treated areas the following day. Since the soil in this locality was rather acid, two trials were prepared. In one trial soil was used without adding ground limestone. In the second trial soil was treated with ground limestone to bring the soil reaction to approximately pH 6.8.

There was no indication in any of the trials that PCNB was toxic to potato plants and that yield had been impaired. In Missaukee County (Table 1), and in Menominee County (Table 2) yields were significantly increased with PCNB. At Ottawa County (Table 2) a non-significant yield reduction was obtained, whereas at all other locations non-significant yield increases followed PCNB treatment.

Scab incidence was consistently reduced (Table 2) (Fig. 2) at a significant level where PCNB was applied at 50 pounds of active material per acre.

Yields were increased significantly with urea-formaldehyde over either PCNB or the control in both trials at Houghton County. Yield reduction in Menominee County was directly related to lack of weed control due to factors beyond the control of the cooperating grower. Urea-formaldehyde stimulated weed growth over and above that in the untreated areas. Furthermore, PCNB seemed to inhibit excessive weed development. Scab incidence was

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[1] Journal Paper Number 2422 of the Michigan Agricultural Experiment Station, East Lansing, Michigan.

[2] Furnished as Terrachlor by the Olin Mathieson Chemical Corporation as 20 percent active pentachloronitrobenzene.

[3] UF-85 furnished by the Allied Chemical and Dye Corporation as 26 percent urea and 59 percent formaldehyde.

Table 1. Potato yields and scab incidence in the Chippewa variety following pentachloronitrobenzene soil treatment, 1955.

Plot location	Soil type and treatment rate[a] (pounds per acre)	Yield cwt/A	Scab incidence (percent): Tubers free from scab	Tubers with less than 5 percent surface scabbed	Tubers with over 5 percent surface scabbed
Leelanau County	Sandy loam				
none		74	11.5	48.0	52.0
PCNB	45	88	47.8	70.8	29.2
PCNB	78	77	49.5	69.4	30.6
PCNB	244	71	75.8	90.2	9.8
LSD 5%		N.S.	25.0	17.9	
1%		--	36.0	25.8	
Missaukee County	Sandy stony loam				
none		87	9.0	31.5	68.5
PCNB	29	81	45.0	61.0	39.0
PCNB	58	76	42.0	54.8	45.2
PCNB	77	95	43.8	58.2	41.8
PCNB	126	75	51.0	73.5	26.5
PCNB	174	105	56.2	71.8	28.2
LSD 5%		19.7	20.5	18.6	
LSD 1%		--	28.4	25.8	

[a]Rates are calculated on the basis of 100 percent active PCNB applied per acre of planted potatoes. In the case of band application rates were increased correspondingly per unit area treated.

FIGURE 1. All-Chem soil treating equipment for application of PCNB.

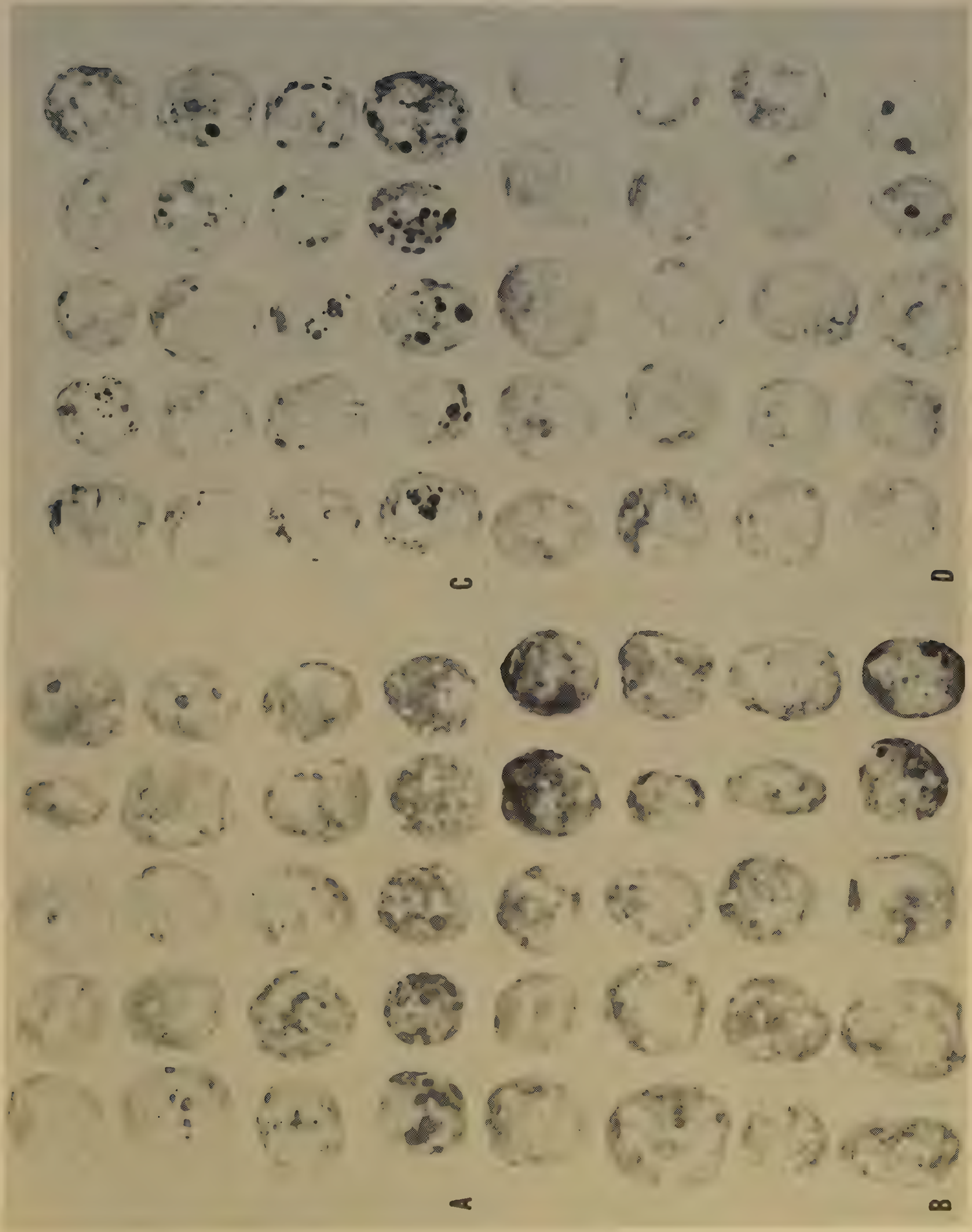

FIGURE 2. Representative tubers from Houghton County plots: A, control plots soil pH 6.8; B, similar plots receiving 150 gallons 1A urea formaldehyde; C, control plots soil pH 5.9; D, PCNB 50 pounds active material per acre.

reduced by urea-formaldehyde to approximately the same degree as with PCNB (Fig. 2).

It seems probable that these materials are potentially useful in potato production in Michigan.

Table 2. Potato yields and scab incidence on Chippewa tubers following various soil treatments at four locations in Michigan, 1958.

Plot location, soil type, soil reaction, and treatment[a]	Yield: Total cwt/A	Yield: Tubers over 1 7/8 in. cwt/A	Yield: Tubers under 1 7/8 in. cwt/A	Scab incidence (percent): Tubers free from scab	Scab incidence (percent): Tubers with less than 5 percent surface scabbed	Scab incidence (percent): Tubers with over 5 percent surface scabbed
Montcalm County						
Sandy loam, pH 6.4						
None	211	189	22	52	84	16
PCNB 50 pounds/A[a]	218	196	22	82	96	4
L.S.D. .05	N.S.	N.S.	N.S.	26	10	10
L.S.D. .01				N.S.	N.S.	13
Ottawa County						
Muck, pH 6.6						
None	296	110	186	19	37	63
PCNB 50 pounds/A[a]	249	210	39	64	86	14
L.S.D. .05	N.S.	62	34	26	34	34
L.S.D. .01		N.S.	N.S.	40	N.S.	N.S.
Menominee County						
Sandy loam, pH 6.6						
None	203	190	13	44	74	26
PCNB 50 pounds/A[a]	259	245	14	86	98	2
Urea-formaldehyde 150 gallons/A[b]	146	134	12	85	94	6
L.S.D. .05	29	29	N.S.	10	4	4
L.S.D. .01	34	34	N.S.	14	6	6
Houghton County						
Sandy loam, pH 6.8						
None	253	228	25	8	32	68
PCNB 50 pounds/A[a]	264	240	26	84	91	9
Urea-formaldehyde 150 gallons/A[b]	311	290	22	70	88	12
L.S.D. .05	32	26	N.S.	10	13	13
L.S.D. .01	N.S.	31		13	18	18
Houghton County						
Sandy loam, pH 5.9						
None	225	209	16	30	61	39
PCNB 50 pounds/A[a]	234	199	35	91	95	5
Urea-formaldehyde 150 gallons/A[b]	286	263	23	88	96	4
L.S.D. .05	24	22	N.S.	8	8	8
L.S.D. .01	37	32		11	11	11

[a]Rates of PCNB calculated in pounds of 100 percent active material per acre.
[b]Urea 26 percent and 59 percent formaldehyde.

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MICHIGAN AGRICULTURAL EXPERIMENT STATION, EAST LANSING

EVOLUTION OF VIRUSES IN ROOTS

C. E. Yarwood

Abstract

If we accept the hypothesis that lack of injury of a host by a parasite is evidence of the evolution of the parasite in that host, then we have a case for the evolution of some plant viruses in roots. Some evidence in support of this hypothesis is reviewed and is contrasted with evidence for the origin of other plant viruses in their insect vectors.

It is a common hypothesis that stable association between host and parasite represents an advanced state of evolution. Stated otherwise, microorganisms have been longer established as parasites of hosts in which they multiply but cause little damage than of hosts in which they cause severe damage.

Andrewes (1) has stated the above hypothesis as "The condition of host-virus tolerance is probably a late adaptation in evolution: open warfare is likely to be the more primitive state." According to Bawden (2) "......and their continued survival is more assured in hosts in which they are harmless or beneficial. Excessive virulence towards a host probably means a newly acquired habit, or that the virus itself has changed to a form that is unlikely to survive. The serious disease outbreaks by which viruses attract attention may be passing events....., ... and it is to tolerant organisms that we should look for the origins of our current pathogens." This point of view, also supported by several others, would favor the hypothesis that some plant viruses have evolved as root parasites, as presented here.

There is little concrete evidence of the basic origin of viruses. Some regard them as derived from more complex organisms by regressive evolution (5). Others emphasize the evidence relating viruses to genes (9). Here we are concerned with relatively late aspects of virus evolution; specifically, what were the immediate ancestors of present plant viruses?

The best evidence relating to the hypothesis proposed is with tobacco necrosis virus (TNV), which is a common parasite in the roots of plants. Smith (12) first pointed out that it was possible to inoculate the leaves of an apparently healthy plant with juice from its own roots and to produce a severe disease. He suggested that TNV might be a transitional stage between a pathogen and a non-pathogen. Several others have confirmed Smith's observation, and Yarwood (16) has suggested that TNV infection of roots may even increase plant growth. With tobacco mosaic virus (TMV), White (15) and Bergmann (3) have reported that infected detached roots grow as rapidly as non-infected roots, and Papasolomontis (11) has indicated that detached TMV-infected roots may even grow more rapidly than non-infected roots, though it is well known that TMV infection of the tops of tobacco and several other plants decreases plant growth. The viruses of tomato bushy stunt, peach yellow bud mosaic, tobacco ring spot, beet ring spot, and beet latent are also believed to increase in seedling roots without any apparent deleterious action on plant growth (17), though each of these viruses may interfere with plant growth if causing an infection of foliage.

Tobacco mosaic virus on tobacco hybrid (Nicotiana tabacum X N. glutinosa) as reported by Fulton (4) is the only case known to me of apparent interference with root growth by a soil-borne virus. This is regarded as a special case, as this same virus in tobacco roots (3, 11, 15), is also the best example of lack of root injury due to virus infection.

It may be argued that infection of roots with virus is synonymous with infection of the entire plant, since most viruses are systemic. There is much evidence against, this, however. One of the most characteristic aspects of TNV is that the virus usually stays in the roots. While Smith (12) noted that TNV was found in tobacco stems in small quantities and without symptoms on a few occasions, he emphasized that it usually remained localized in the roots without symptoms. Fulton (4), Harrison (6), and Lucas (8) have also emphasized the limited movement of viruses from roots to tops, so that in the field the virus may frequently be found only in roots. With most foliage infections except TNV, the virus soon moves from the tops to the roots, but for infections originating in the root, the virus moves slowly or not at all to the tops. Even this slow movement of the virus from roots to tops under experimental conditions may be much faster than occurs in nature where plants grow under less favorable conditions, though

no evidence in support of this is known.

If some present plant viruses evolved from root parasites it is likely that they came from the soil, as soil is the greatest known reservoir of microorganisms, including bacteriophages. Many bacterial and fungus pathogens of higher plants also likely evolved from soil inhabitants. The best supporting evidence known to me is with Pseudomonas tabaci and P. angulatum. Valleau et al. (14) have shown that these bacteria occur on the roots of tobacco and other plants without causing any apparent injury, but when these bacteria are introduced into tobacco leaves they cause the serious diseases of wildfire and angular leaf spot. The situation with P. tabaci and P. angulatum is thus closely analogous to the situation with TNV as described briefly above.

The only comparable hypothesis of the origin of plant viruses is that some of them have evolved from insect viruses (10). Here the principal evidence used is that infection of the insect vector causes less injury than infection of the plant by these same viruses. Of the five viruses cited by Maramorosch (10), only one is known to cause symptoms in the insect vector, and even in this case no macroscopic injury to the insect is recognized. Only one vector of a plant virus is known to be injured apparently as a result of carrying the virus (7), and this peach yellow leaf roll virus is not yet known to multiply in the insect.

The factual evidence for the origin of some plant viruses in roots is therefore basically similar to the evidence for the origin of other plant viruses in insects. In both cases, it is based principally on the lesser injury to the hypothetical original host or host part than to the host or plant part that shows symptoms. The two hypotheses are not mutually exclusive. Actually there are no overlapping examples, and one hypothesis might apply to one set of viruses and the othe hypothesis to another set of viruses. Further, there is limited circumstantial evidence, given in part by Smith (13), that none of the viruses cited here as having probably evolved as root parasites has an insect vector.

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY

A SYSTEMIC DISEASE OF BEANS CAUSED BY A TOBACCO NECROSIS VIRUS[1]

John J. Natti

Summary

A naturally occurring systemic disease of bean (Phaseolus vulgaris) observed at Geneva, New York was found to be caused by a tobacco necrosis virus. Symptoms in experimentally inoculated plants indicated the disease to be closely related to, if not identical with, stipple streak, a virus disease of bean that has been observed only in Holland. All of 67 varieties of P. vulgaris inoculated in greenhouse tests became systemically infected. No symptoms were observed on, and the virus was not recovered from, inoculated leaves of P. coccineus, Pisum sativum, Lotus corniculatus, Medicago sativa, Melilotus alba, Trifolium hybridum, T. incarnatum, T. pratense, and T. repens. In pasteurized soil infested with the virus, 13 percent of the bean plants (California Red Kidney) from direct seeding, and 52 percent of the plants from transplanted bean seedlings developed systemic infection.

Tobacco necrosis viruses in nature usually occur in the roots of plants without causing symptoms either in the roots or in the above-ground parts of the plants (9). The presence of the virus in the roots can be demonstrated by the development of localized necrotic spots in the leaves of plants inoculated with juice pressed from the roots. The natural occurrence of tobacoo necrosis virus has been reported in a relatively few plant species in comparison with the wide range of susceptible plant species indicated by foliage inoculation tests (6). Only a few plant species become systemically infected. The systemic infection of Primula obconica caused by a tobacco necrosis virus has been observed in England (1, 8) and in the United States (7). A systemic disease of tulip in Holland was discovered to be caused by a tobacco necrosis virus (5). A similar disease of tulip has been observed in the United States (3). Stipple streak, a serious virus disease of beans in Holland (4, 10), was found to be caused by a tobacco necrosis virus (2). In 1957, at Geneva, New York, two bean plants in the greenhouse and six plants of a breeding line of beans in a field plot were infected with a disease which appeared to be of bacterial nature. Host range and transmission tests indicated that the disease was caused by a tobacco necrosis virus. This is the first report of the natural occurrence in the United States of a systemic infection of bean caused by a tobacco necrosis virus.

SYMPTOMS OF SYSTEMIC INFECTION OF BEAN PLANTS

Symptoms of systemic infection of bean plants from natural inoculations in the field and greenhouse were similar. Usually one side of the plant was severely infected while the other side appeared to be normal. A narrow dark-brown band of necrotic tissue extended along the stem and the petioles of some of the lower leaves. Symptoms in the leaves consisted of irregular areas of varying size in which the vascular tissues were dark-brown (Fig. 1). Severely infected leaves shrivelled and later abscised. Dark-brown necrotic tissues extended along the sutures of some of the pods. Dark-brown areas suggestive of ringspot lesions occurred in the pods, but the seeds were not infected. The necrotic tissues were dry and firm.

In the greenhouse, small brown necrotic spots developed in inoculated bean leaves 18 to 24 hours after inoculation. The virus spread from the necrotic spots to surrounding tissues to produce areas with darkened veins. The virus spread from the inoculated leaves to the stem and to upper uninoculated leaves. Symptoms in these invaded leaves consisted of irregular areas containing a network of darkened veins. The darkening of the vascular tissues was usually preceded by a yellowing of the foliage in the infected area. No mottling of uninvaded tissues was observed. The pulvini of the petioles of some of the trifoliate leaves was so se-

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1 Approved by the Director as Journal Paper No. 1154, New York State Agricultural Experiment Station, Geneva, New York.

FIGURE 1. Infected areas in trifoliate leaf of a bean plant invaded by virus from inoculated primary leaf. Note network of darkened veins in lesions, and the severe necrosis of the pulvini.

FIGURE 2. Ringspot-like lesions on leaf of Nicotiana rustica inoculated with virus causing systemic infection of bean.

verely infected (Fig. 1) that the tissues shrivelled and served as a barrier to the movement of the virus from or into the leaflets.

FOLIAGE INOCULATION AND SOIL TRANSMISSION TESTS

Methods

All tests were conducted in the greenhouse at temperatures of 65° to 75° F. Systemic infection of beans occurred at these temperatures, whereas only scattered localized lesions developed in inoculated leaves at temperatures of 80° to 90° F.

Plants for the foliage inoculations were grown in pasteurized greenhouse compost in 4-inch pots. The plants were inoculated by rubbing the leaves with a cheesecloth pad that had been dipped in juice pressed from leaves of infected beans (California Red Kidney). Carborundum was used as an abrasive. In the soil transmission tests, dried infected bean leaves were ground into a coarse powder and one volume of this powder was mixed thoroughly with 800 volumes of pasteurized greenhouse compost. In some tests seeds were planted directly in the virus-infested soil. In other tests seedlings that had been germinated in quartz sand were transplanted in the virus-infested soil.

In the foliage inoculation tests the inoculated leaves and upper uninoculated leaves were tested for the presence of the virus by inoculating bean leaves with juice pressed from the test plants. The presence of the virus in the roots was determined by inoculating bean leaves with juice pressed from the roots of the test plants. The roots were thoroughly washed prior to extraction of the juice, in order to remove soil and plant debris which might contain the virus.

Results

Foliage Inoculation Tests: The following varieties of beans developed systemic infection from inoculated primary leaves: Antwerp, Black Turtle Soup, Black Valentine, Black Wax, Bountiful, Bountiful Canner, California Red Kidney, Chevert Stringless, Cherokee Wax, Columbia, Commodore, Contender, Cranberry, Davis Stringless Wax, Dixie Belle, Dwarf Horticultural Long Pod, Emerson 15, French Horticultural, Giant Stringless, Golden Gem, Horticultural, Hyscore, Idaho Refugee, Improved Full Measure, Improved Tendergreen, King Green, Kinghorn Wax, Large White Marrow, Logan, Low's Champion, Mammoth Horticultural, Marrow, Michelite, New Improved Tendergreen, Pearl Green, Pencil Pod, Plentiful, Processor, Puregold Wax, Ranger Red Mexican, Refugee, Refugee Stringless, Seminole, Slendergreen, Soldier, Streamliner, Stringless Blue Lake, Stringless Green Pod, Stringless Horticultural, Striped Halfrunner, Style's Blue Lake, Sulphur, Supergreen, Sure Crop Black Wax, Tenderbest, Tendergreen, Tenderlong, Tennessee Green Pod, Topcrop, Topmost, Topnotch Golden Wax, Unrivalled Wax, Vermont Cranberry, Wade, White Halfrunner, White-seeded Refugee, and Yellow Eye. Manchu, the one variety of *Glycine max* tested, also became systemically infected.

Localized necrotic lesions developed on inoculated leaves of *Phaseolus lunatus* varieties Fordhook 242 and Henderson's bush lima. The virus was not recovered from the inoculated leaves.

No symptoms were observed on, and virus was not recovered from, inoculated leaves of the following plants: *Phaseolus coccineus* var. Scarlet Runner and White Dutch Runner bean; *Lotus corniculatus* var. Viking E; *Medicago sativa* var. Ranger; *Melilotus alba*, sweet clover; *Trifolium hybridum*, alsike clover; *T. incarnatum*, crimson clover; *T. pratense*, medium red clover and mammoth red clover; *T. repens*, ladino and white clover; and *Pisum sativum* var. Bonneville, Laxton's Progress, Perfection, Profusion, and Stratagem.

Localized necrotic lesions developed in inoculated cotyledons and leaves of the following plants: *Cucumis sativus* var. Boston Pickling, Chicago Pickling, Early Fortune, Marketer, Niagara, Ohio M. R. Pickling, and Straight Eight; *Cucurbita maxima* var. Blue Hubbard, Buttercup, Green Hubbard, Royal Acorn, and Small Sugar; *Cucurbita moschata* var. Butternut; and *Cucurbita pepo* var. Banana Squash, Conn. Field Pumpkin, Early Crookneck, Straightneck, Table Queen, UConn, White Bush Scallop, Winter Luxury, and Zucchini. The virus was not recovered from uninoculated leaves of these plants and no evidence of systemic infection was observed.

Local necrotic spots developed in inoculated leaves of *Beta vulgaris* var. Detroit Dark Red; *Capsicum frutescens* var. California Wonder; *Datura stramonium*; *Gomphrena globosa*; *Ipomoea tricolor* var. Heavenly Blue; *Lycopersicum esculentum* var. Bonny Best; *Nicotiana rustica*; *Petunia hybridum* var. Comanche; *Santivalia procumbens*; *Spinacia oleracea* var. Heavy Pack; and *Zinnia elegans* var. Blaze. Virus was not recovered from uninoculated leaves of these plants.

A very limited spread of the virus from lesions in leaves of *Nicotiana rustica* resulted in the development of a zone of infected tissues surrounding the initial site of infection (Fig. 2).

Symptoms were not observed on, nor was virus recovered from, inoculated leaves of *Calendula officinales*, *Cichorium endivia*, and *Solanum tuberosum* (USDA Seedling 41596).

Soil Transmission Tests: Thirteen of 100 bean plants (California Red Kidney) originating from seeds planted in virus-infested soil, and 52 of 100 bean plants from seedlings germinated in quartz sand and transplanted in the virus-infested soil developed lesions on the stems or foliage. Virus was recovered only from the leaves with symptoms. However, virus was recovered from roots of plants that had no foliage symptoms as well as from those with symptoms.

In other tests in which seedlings were transplanted in virus-infested soil, virus was recovered from the roots of *Beta vulgaris* var. Detroit Dark Red; *Capsicum frutescens* var. California Wonder; *Gomphrena globosa*; *Lycopersicum esculentum* var. Bonny Best; *Nicotiana rustica*; and *Petunia hybridum* var. Comanche. None of the plants developed symptoms in either the foliage or roots. Virus was not recovered from the foliage of any of these plants.

CHARACTERISTICS OF THE VIRUS

In determining certain physical characteristics of the virus, bean plants (California Red Kidney) were utilized both as a source of the virus and as test plants.

Thermal inactivation. Two ml of freshly extracted juice from infected bean leaves was heated for 10 minutes in sealed thin-walled glass tubes. The tubes were immediately cooled in cold running water after completion of the heating period. In two of the three tests conducted, the virus was completely inactivated in juice heated at 90° C, but active virus in low concentration was present in one test at this temperature. In each of three tests, on the basis of local lesion counts, the active virus titre was reduced by about 90 percent by treatments at 80° C.

Resistance to aging. Undiluted juice from infected bean leaves was maintained in a stoppered test tube at 22° to 25° C. Bean leaves were inoculated at 2-day intervals with this juice. There was no indication of loss of infectivity by the virus during 10 days of storage.

The virus in leaves that had been stored in a dried condition at 22° to 25° C for 316 days still remained highly infective.

Dilution end-point. Juice from bean leaves infected for 4 days was diluted with distilled water to obtain a series of dilutions ranging from $1:10^2$ to $1:10^6$. Scattered local lesions developed on bean leaves inoculated with dilutions of $1:10^5$ to $1:10^6$.

Immunological reactions. The presence of the virus in the roots conferred no protection on the foliage of the plants. Infection of the primary leaves of bean plants from experimental inoculations did not provide protection against subsequent infection of the inoculated leaves or of upper uninoculated leaves.

DISCUSSION

The occurrence of the virus in roots of normal-appearing plants, the symptoms produced on foliage of experimentally inoculated plants, and the physical properties of the virus served to identify the causal virus of the systemic infection of beans described in this report as a tobacco necrosis virus. Tobacco viruses are unique in that they occur in nature in the roots of many plants without causing symptoms either in the roots or in the foliage. However, the virus in juice pressed from the roots will cause the formation of localized necrotic lesions in inoculated leaves of a wide range of plant species. The systemic disease of beans discussed in this report appears to be closely related to, if not identical with, stipple streak, a serious disease of beans in Holland (4, 10) caused by a strain of the Rothamsted tobacco necrosis virus (2).

The origin of the virus in the greenhouse compost and the field at Geneva is not known. It is possible that the field infection resulted from virus transferred from the greenhouse on seeds or in plant debris, since the infected plants originated from seeds produced in the greenhouse.

Although this disease does not appear to be of economic importance at this time, its potential seriousness should not be disregarded. Since all commercial bean varieties tested were highly susceptible, control by planting resistant varieties is not possible. Because the virus can overwinter in the soil or in crop debris, beans should not be planted in fields in which the disease was observed the previous year. On the basis of the insusceptibility of various clovers to the virus in this investigation, a crop rotation schedule in which clovers precede beans offers promise as a method of control.

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DEPARTMENT OF PLANT PATHOLOGY, NEW YORK STATE AGRICULTURAL EXPERIMENT STATION, GENEVA, NEW YORK

FATE OF STRAWBERRY PLANTS INFECTED WITH ASTER YELLOWS VIRUS IN NURSERY BEDS

Norman W. Frazier and Harold E. Thomas[1]

The present authors[2] observed that strawberry plants infected with aster yellows virus in the field appeared to die within a short period of time. The course of the disease in individual plants infected under natural conditions had not been followed and it was not known whether diseased plants found in the fruiting fields in Central California had been infected there or in the nursery beds of Northern California where most California strawberry plants are produced.

The incidence of aster yellows in nursery beds varies from year to year. Usually it is negligible, but during some years has been of concern. Most infected plants die or are rogued out by the time the nursery plants are dug, but many may still remain. Since, as a result of an infection, the virus spreads to all daughter plants attached to a mother plant and the infected plants may be dug with the normal plants and set in the new fruiting fields, it was desirable to know the fate of such infected plants.

On December 1, at the start of commercial digging operations, 453 aster yellows-infected nursery plants were dug by hand. All of the living daughter plants of each infected mother-daughter clone were taken. Plants of infected clones were stunted, chlorotic with reddish older leaves, flattened in appearance and leaves cupped. Many plants were dead or severely weakened. Eight commercial strawberry varieties were represented; the variety Lassen and seven varieties developed by the Strawberry Institute bearing code designations. The plants were stored and planted in a manner comparable to normal commercial practice. They were set out on April 22 in a test plot in a fruiting area near Gilroy. At the time of planting it was estimated that if the diseased plants had been mixed with normal plants in a commercial nursery operation, approximately 5 percent of them would have been discarded as culls in the sorting and packaging.

On June 11, seven weeks after planting, 398 plants were dead, 50 were obviously diseased, and 5 plants were symptomless. The 5 plants were the only ones alive of 110 of one variety and, since they remained normal for the duration of the test, it is probable that they represented healthy plants dug in error. All plants of four varieties were dead. The variety Lassen, with 44 percent survival of plants, appeared more tolerant of the virus than any other.

On September 4 only 10 plants were alive, one that was very weak and nine that had recovered to the extent that they were fairly vigorous, had formed stolons and daughter plants, and lacked diagnostic symptoms of aster yellows although they did not appear entirely normal.

Later, on October 17, nine plants remained, eight of which had again developed obvious symptoms of disease. One plant, a mother with only very mild symptoms and with two attached rooted daughters that appeared symptomless, was brought to the glasshouse for further observation. Within a period of 6 weeks the mother and both daughters developed clear symptoms of aster yellows.

From the results, it seems evident that most aster yellows-infected strawberry nursery plants die shortly after being planted out in fruiting fields. A very few may survive a longer period and even show some degree of recovery. Under the conditions of this test, recovery was temporary rather than permanent.

DEPARTMENT OF ENTOMOLOGY AND PARASITOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY, AND THE STRAWBERRY INSTITUTE OF CALIFORNIA, MORGAN HILL

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[1] Respectively, Associate Entomologist, California Agricultural Experiment Station, Berkeley and Director, The Strawberry Institute of California, Morgan Hill.

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CONTROL OF GREY MOLD ON STRAWBERRIES

P. M. Miller and E. M. Stoddard

Abstract

Lengthening the time between the last spray and harvest increased the severity of grey mold. Thiram gave the best control, followed by dichlone, and captan was least effective. Thiram reduced post-harvest rot by two-thirds at 16 days after spraying, but there was practically no control 21 days after spraying.

Grey mold of strawberries, caused by Botrytis cinerea Pers., is a perennial threat to the strawberry grower. If moist weather prevails after bloom, 25 to 50 percent of the fruit may be rotted. Previous reports by the authors (1, 2) included a review of work by others on grey mold control. The work reported here studied the effect of timing and number of applications on control of pre-harvest grey mold infections and the influence of pre-harvest sprays on post-harvest grey mold.

Laboratory tests have shown that the calyx was the most susceptible part of the plant to infection by B. cinerea. This suggested that pre-blossom sprays should reduce pre-harvest grey mold infection. Previous work has shown that thiram and dichlone had a longer residual effect than other fungicides tested and indicated the desirability of these materials where a minimum number of sprays is important and it also indicated the probability that post-harvest rotting could be reduced.

The experiments were conducted on two varieties of strawberries, Stelemaster and Sparkle, and each variety will be discussed separately.

STELEMASTER

In this experiment 10-foot plots of Stelemaster, replicated four times, were sprayed with Thylate (65% wettable thiram), Phygon XL (50% wettable dichlone) and Orthocide 406 (50% wettable captan). The materials were used at a concentration of 3 pounds, 3/8 pounds and 6 pounds per hundred gallons respectively. The Thylate and Phygon plots were divided into three groups. Group 1 had only one pre-bloom spray on May 10. Group 2 had two pre-bloom sprays on May 10 and May 22. Group 3 had two pre-bloom sprays on May 10, 22 and another spray on June 4 when primary fruits were half grown. Group 4 had three sprays of captan on the same dates as the plots in group 3.

Control of pre-harvest infection was determined by a count of all berries infected with Botrytis from each plot on June 20, this date being 41 days from the last spray in group 1, 29 days in group 2 and 16 days in group 3. (Table 1). It will be noted that in group 1 there was no control and that an increasing number of sprays gave increased control in groups 2 and 3. Comparison of the effectiveness of the three materials on the basis of three sprays shows that Thylate gave the best control.

The effect of spraying on post-harvest grey mold infection was determined from a random sample of 2 quarts of sound berries picked on June 20 and 25 from each plot in groups 3 and 4. The percent of grey mold on the first picking was determined at 1 and 4 days after harvest. On the second picking counts were made only at 1 day after harvest as all berries were rotted on the fourth day (Table 2). These data show that Thylate was the most effective in reducing post-harvest rot 16 days after the last spray. There was little difference between treatments or between sprayed and check plots at the last picking, indicating that at 21 days after the last spray the value of all treatments was practically nil.

SPARKLE

In this test 50-foot plots of Sparkle strawberries, replicated four times, were sprayed May 1, 10, and 20, June 2 (blossom spray) and 12. The fungicides and concentrations were the same as those used in the Stelemaster plots except that 1 1/2 pints of a sticker-spreader per 100 gallons were added to the Orthocide 406.

Control of pre-harvest infections was determined by picking all the visibly infected berries in the plots on July 2 and 10, which was 18 and 28 days after the last spray (Table 1). Thiram

Table 1. Effect of timing and number of sprays on pre-harvest grey mold rot.

Fungicide (amount per 100 gallons)	Dates of application	Number of rotted berries per 10-foot row: Stelemaster plots (June 20)	Sparkle plots (July 2)	Sparkle plots (July 10)
Phygon XL 3/8 pound	May 10	103		
	May 10, 22	89		
	May 10, 20, June 4	39		
	May 1, 20, June 2, 12		27	55
Thylate 3 pounds	May 10	115		
	May 10, 22	63		
	May 10, 22, June 4	26		
	May 1, 10, 20, June 2, 12		24	46
Orthocide 406 6 pounds	May 10, 22, June 4	55		
Orthocide 406-6 pounds + 1 1/2 pints sticker-spreader	May 1, 10, 20, June 2, 12		51	64
Unsprayed		92	49	58

Table 2. Influence of pre-harvest sprays on post-harvest grey mold rot of Stelemaster strawberries.

Fungicide (amount per 100 gallons)[a]	Date picked	Date of last spray	Percent grey mold[b] and date of count: June 21	June 24	June 26
Phygon XL 3/8 pound	June 20	June 4	3.5	50.8	
	June 25				23.4
Thylate 3 pounds	June 20	June 4	1.0	27.9	
	June 25				18.9
Orthocide 406 6 pounds	June 20	June 4	3.7	42.9	
	June 25				20.5
Unsprayed	June 20		6.7	69.3	
	June 25				27.9

[a]Sprayed May 10, 22, and June 4.

[b]Average of 8 quarts, 2 quarts from each plot.

gave the best control at both pickings. (Control by all materials decreased with time and was almost non-existent by July 10, 28 days after the last spray). The sticker-spreader added to Orthocide 406 completely destroyed its fungicidal value and there were slightly more infected berries in these plots than in unsprayed plots.

Berries were retained from the first picking on July 2 for post-harvest grey mold counts. Very heavy and general occurrence of leak, caused by Rhizopus nigricans, within 48 hours after picking completely obscured all grey mold infection. None of the spray treatments controlled the Rhizopus.

Yield data were taken at the second picking and at the maximum yield period. There was no reduction in yield or fruit size by any treatment. The thiram plots had a slightly higher yield than other plots.

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DEPARTMENT OF PLANT PATHOLOGY AND BOTANY, CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN

BIPHENYL-INDUCED VARIATIONS IN CITRUS BLUE MOLD

Paul R. Harding, Jr.[1]

Summary

Resistant and semiresistant strains of citrus blue mold (Penicillium italicum Wehmer) were produced by continued exposure to biphenyl. Resistant strains in the presence of very high concentrations of biphenyl grew at the normal rate and produced white mycelium which soon developed into typical heavily sporulating colonies of glaucous color. Semiresistant strains exposed in the same manner grew at about half the normal rate and were of two types, one which produced white nonsporulating colonies and the other which produced sporulating colonies of the usual glaucous blue color. All strains obtained in this manner proved virulent when inoculated into lemons.

Strains of blue mold that were nonresistant to biphenyl produced dark red nonsporulating colonies when in the presence of high concentrations of biphenyl. Some of these strains turned red in about 3 days, while others required 2 or more weeks of exposure to develop this color.

Neither sodium orthophenylphenate nor limonene played any part in the development of these variants of blue mold. A high concentration of limonene neutralized part of the effectiveness of biphenyl in retarding growth and preventing sporulation, but the small amount (1.0 percent) used in biphenyl paper made no difference.

INTRODUCTION

The present study was made to investigate the frequent appearance of a dark red mold on lemons (Fig. 1) in storage experiments at the Sunkist Field Laboratory, Ontario, California in the summer of 1957. On fruit held at 55° F in nonvented cartons containing biphenyl paper, the mold developed without sporulation and appeared "Vandyke Red"[2], owing to the presence

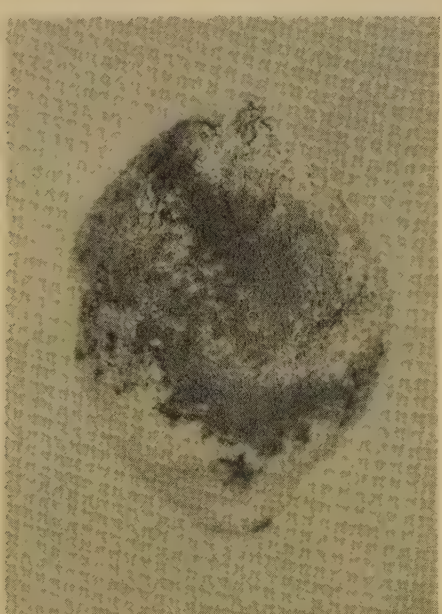

FIGURE 1. Red variant of Penicillium italicum on lemon.

[1]Mycologist, Biological Sciences Branch, Agricultural Marketing Service, United States Department of Agriculture, Pomona, California.

[2]Color designations referred to in this paper are described by Ridgway (2).

Table 1. Treatments used on inoculated and uninoculated lemons and corresponding colony color developed by biphenyl-nonresistant (normal) strains of Penicillium italicum.

Treatment number	Material used	Color of mold colony
Inoculated:		
1	Sodium orthophenylphenate	Blue
2	Sodium orthophenylphenate and wrapped in paraffined mineral-oil paper	Blue
3	Sodium orthophenylphenate and biphenyl in dishes	Red
4	Sodium orthophenylphenate and wrapped in biphenyl paper	Red
5	Biphenyl in dish	Red
6	Limonene in dish	Blue
7	Biphenyl plus limonene (1:1) in dishes	Blue
8	Biphenyl paper without limonene	Red
9	Biphenyl paper with 1 percent limonene	Red
10	Control (no orthophenylphenate treatment)	Blue
11	Control (no orthophenylphenate treatment) wrapped in paraffined mineral-oil paper	Blue
Uninoculated:		
12-22	Treatments same as above series	No molding

of abundant intracellular pigment. When isolated in pure culture on nutrient agar and grown in the absence of biphenyl, the fungus was determined to be blue mold (Penicillium italicum Wehmer).

California lemon-packing houses recently adopted the use of sodium orthophenylphenate in the postharvest washing solution to reduce fungal decay during storage. Another change was the addition of limonene as a deodorant in biphenyl paper[3]. The original purpose of this investigation was to find out if these changes in packing house methods were responsible for the development of the red variant of P. italicum on lemons stored in the presence of biphenyl. However, the course of the work led to some interesting findings concerning biphenyl-resistant, -semiresistant and -nonresistant strains of P. italicum.

MATERIALS AND METHODS

The work was performed in three phases: 1) to determine whether the use of sodium orthophenylphenate could be responsible for the development of the red variant of P. italicum on lemons stored in the presence of biphenyl; 2) to determine whether biphenyl, limonene, or biphenyl plus limonene could be responsible for this variant; and 3) to study the development of biphenyl-resistant strains of P. italicum. The treatments are outlined in Table 1.

Testing Sodium Orthophenylphenate

An experiment was performed to determine whether sodium orthophenylphenate could be responsible for the development of the red variant of P. italicum on lemons stored in the presence of biphenyl. In a typical packing house, lemons were subjected for 3 minutes to 70° F, alkaline (pH 12) washing solution of the following composition: 34 percent stock solution of sodium orthophenylphenate, 32 gallons; trisodium phosphate, 80 pounds; sodium hydroxide, 25 pounds; water, 1500 gallons. After being washed in this solution, the lemons were spray-rinsed in plain water. Lemons treated in this manner were compared with nontreated lemons by inoculating the fruit of the respective lots with P. italicum and exposing them to high concentrations of biphenyl. Ten lemons were used per 12-liter glass chamber and placed in close association with biphenyl by wrapping them individually in sheets of biphenyl paper lacking limonene (Table 1, treatment 4), or by placing them on perforated shelves immediately above a 9.6 square-inch surface of caked biphenyl (Table 1, treatment 3). The caked biphenyl

[3] One sheet 17 x 11 inches contains 2.35 grams of biphenyl in a mixture of biphenyl 50 parts, paraffin 25 parts, mineral oil 24 parts, and limonene 1 part.

was prepared in a Petri dish by melting 15 grams in an electric oven at 175° F and cooling slowly. The chambers used here were the type ordinarily used as chemical desiccators with perforated porcelain shelves and tubulated lids; the lid tube served as a vent and was plugged with cotton. Controls consisted of treated and nontreated lemons that were inoculated but held in chambers lacking biphenyl (Table 1, treatments 1 and 10). Another set of controls consisted of treated and nontreated lemons wrapped in paper containing paraffin and mineral oil but no biphenyl (Table 1, treatments 2 and 11). This experiment was conducted in a 60° room.

Testing Biphenyl and Limonene

Another experiment was performed to test the effectiveness of biphenyl, limonene, and biphenyl plus limonene in the development of the red mold on lemons. Lemons were inoculated with P. italicum and held in chambers which contained one Petri dish with 15 grams of caked biphenyl or one with 15 grams of limonene, or one dish of each of these substances (Table 1, treatments 5, 6, and 7). Some inoculated specimens were wrapped in biphenyl paper with 1.0 percent limonene and some without limonene (Table 1, treatments 8 and 9). Controls consisted of inoculated lemons not exposed to biphenyl or limonene, some of which were wrapped in paraffined mineral-oil paper and some of which were unwrapped (Table 1, treatments 10 and 11). To investigate this matter further, cultures of P. italicum were started on nutrient agar (H-24)[4] in 250-ml Erlenmeyer flasks. The flasks were placed in an inverted position on the perforated shelves of the chambers so that the open mouths of the flasks were immediately above the following treatments: one chamber with a Petri dish containing 15 grams of caked biphenyl, one containing a Petri dish with 15 grams of limonene, one containing a Petri dish of each of these substances, one containing a 9.6 square-inch disk of biphenyl paper with 1.0 percent limonene, and one containing a disk of biphenyl paper without limonene. Cultures of the mold in Erlenmeyer flasks also were kept in chambers containing no chemical. Growth rates in millimeters per 24 hours were determined by averaging daily increments in colony diameter. This experiment was conducted in a room maintained at 60° F.

As a special check on the condition of the lemons used in this work, uninoculated specimens with and without the respective treatments (Table 1, treatments 12-22) were held under the same conditions as in the previously described experiments.

Biphenyl-resistant Strains

In developing and studying biphenyl-resistant strains of P. italicum, cultures derived from single-spore isolations were exposed to a high atmospheric concentration of biphenyl. The inverted Erlenmeyer-flask technique previously described was found to be a satisfactory method of exposure, but another method also was employed which proved superior in effectiveness and convenience. In this method H-24 agar in Petri dishes was inoculated in the center with the mold and the lid replaced with one containing 15 grams of caked biphenyl. These cultures were placed in a glass chamber of the type previously described and kept in a room maintained at 60° F.

RESULTS AND DISCUSSION

Lemons, either treated or not treated with sodium orthophenylphenate, exhibited the nonsporulating red-mold variant of P. italicum 3 to 4 weeks after inoculation with a pure culture of biphenyl-nonresistant (normal) strain when held in close association or in contact with biphenyl (Table 1, treatments 3 and 4). They exhibited typical sporulating blue mold in the absence of biphenyl (Table 1, treatments 1, 2, 10, and 11). The color of the mycelium ranged from "Vandyke Red" in specimens wrapped in biphenyl paper without limonene to "Corinthian Red" on specimens over dishes of biphenyl (Table 1, treatments 3 and 4). Treated and nontreated specimens wrapped in paper containing paraffin and mineral oil but no biphenyl, as well as those that were unwrapped, developed typical blue mold in 2 to 3 weeks (Table 1, treatments 1, 2, 10, and 11).

Lemons exposed to dishes of limonene, or the high proportion of limonene with biphenyl (1:1 ratio), did not develop the nonsporulating red-mold variant. Like the unexposed speci-

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[4] $FeCl_2 . 4H_2O$ 0.1 g., $ZnCl_2$ 0.1 g., $MgSO_4 . 7H_2O$ 0.5 g., KH_2PO_4 0.5 g., $Ca(NO_3)_2 . 4H_2O$ 0.5 g., sodium citrate 0.5 g., yeast extract 2.0 g., neopeptone 2.0 g., sucrose 40.0 g., agar 20.0 g., distilled water 1000.0 ml.

mens, they developed typical blue sporulating mold in 2 to 3 weeks after inoculation (Table 1, treatments 6, 7, and 10). Lemons exposed to dishes of biphenyl alone developed "Corinthian Red" nonsporulating mycelium in 3 to 4 weeks (Table 1, treatment 5). Lemons wrapped in biphenyl paper with 1.0 percent limonene or without limonene developed "Vandyke Red" nonsporulating mycelium in 3 to 4 weeks (Table 1, treatments 8 and 9).

Cultures of P. italicum on nutrient agar in Erlenmeyer flasks inverted over limonene, or 1:1 biphenyl and limonene, did not develop the red-mold variant; they developed white mycelium which produced an abundance of glaucous blue spores after about 5 days in the former treatment and about 2 weeks in the latter. Cultures exposed to biphenyl alone or biphenyl paper with or without 1.0 percent limonene (50 parts biphenyl to 1 part limonene) developed "Vandyke Red" nonsporulating colonies of restricted growth in about 3 days. Exposures were started 24 hours after inoculation. Measurements were started 48 hours after the beginning of exposure. Cultures exposed to no treatment sporulated from the outset to produce glaucous blue colonies that increased in diameter at an average rate of 7.8 mm per 24 hours at 60° F. By way of comparison for the same strain of mold, cultures exposed to limonene increased 7.5 mm per 24 hours, those exposed to high concentrations of both biphenyl and limonene had a growth rate of 2.5 mm per 24 hours, while the diameter of those exposed to caked biphenyl alone or biphenyl paper with or without 1.0 percent limonene increased about 1.0 mm per 24 hours.

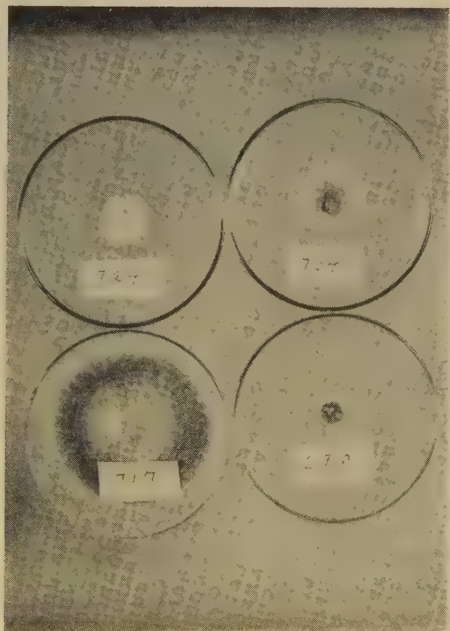

FIGURE 2. Different strains of Penicillium italicum after 2 weeks of exposure to biphenyl: 717, resistant; 724 and 704, semiresistant; 698, nonresistant.

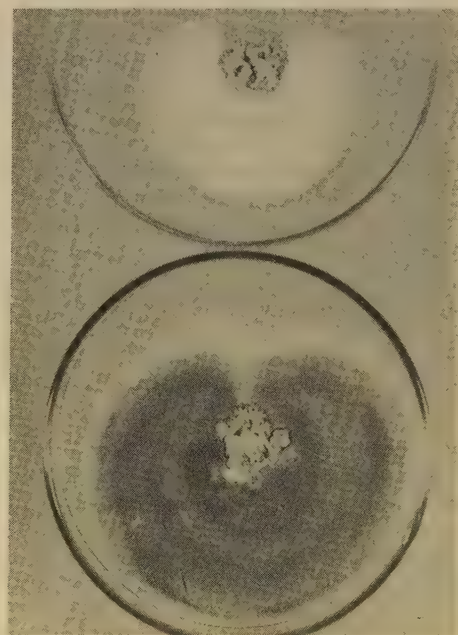

FIGURE 3. Two strains of Penicillium italicum that developed resistance after 20 days of exposure to biphenyl: the white strain (nonsporulating) semiresistant, and the colored strain (sporulating) resistant.

During the previously described part of this work, some resistant strains of P. italicum arose on lemons and in agar cultures and overgrew the parent colonies under the selective influence of biphenyl. These strains were studied in comparison with normal (sensitive) strains using Petri-dish cultures (H-24 agar) with caked biphenyl in the lids. In this technique the agar was inoculated in the center and after 24 hours the plain lid was replaced with one containing caked biphenyl. Growth measurements were started 48 hours after the beginning of exposure. When exposed to the high atmospheric concentration of biphenyl provided by this technique and held at 60°, nonresistant strains grew very slowly (0.13-0.44 mm per 24 hours), did not sporulate, and turned from white to dark red after 3 days to 2 weeks (698, Fig. 2).

Resistant strains grew at the normal rate (6.2-7.6 mm per 24 hours), developing white mycelium which soon sporulated to become glaucous blue in color (717, Fig. 2). Semiresistant strains had intermediate growth rates (1.1-3.25 mm per 24 hours) and were of two types. One produced white nonsporulating colonies (724, Fig. 2) and another produced white colonies that soon sporulated to become glaucous blue (704, Fig. 2).

Resistant strains of P. italicum arose in some cases after as little as 1 to 2 weeks of exposure to very high concentrations of biphenyl. This resistance was acquired, not by the whole colony, but by some internal portion which proceeded under the selective influence of biphenyl to grow out of one side and then to overgrow the parent colony (Fig. 3). In some cases this acquired resistance was evidently the result of genetic mutation. Herein the change was sudden, constant, and permanent, that is, the biphenyl-nonsensitive strain suddenly arose, grew at a fixed rate, and did not lose its resistance upon return to an environment lacking biphenyl. In other cases the acquired resistance was not permanent, but emerged through heterogenous physiological adaptation plus selection wherein some internal portion responded without genetic change and overgrew the parent colony. This was evidenced in that the induced property disappeared rapidly on return to an environment lacking biphenyl. Homogeneous physiological adaptation wherein all of the cells of the colony gradually develop resistance to the inducing agent was not encountered. Bryson and Demerec (1), Stanier (3), and Szybalski and Bryson (4) have discussed the mechanisms by which microorganisms acquire resistance. In the development of genetic adaptation, it was not possible to distinguish between induced and spontaneous mutations. Resistance could develop as a result of selection of either. None of the strains obtained in this manner showed any decline in virulence when inoculated into lemons.

Resistant strains of the citrus green mold (Penicillium digitatum Sacc.) can be produced by biphenyl exposure just as easily as the citrus blue mold (P. italicum), although in the former the preliminary development of red mycelium did not occur.

Recently it has been observed that citrus blue mold will produce red nonsporulating mycelium in the presence of sublethal concentration of chlorine. Experimentation has shown that these same strains will also turn red in the presence of biphenyl. No further investigation of this peculiarity has yet been made.

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UNITED STATES DEPARTMENT OF AGRICULTURE, AGRICULTURAL MARKETING SERVICE, BIOLOGICAL SCIENCES BRANCH, QUALITY MAINTENANCE AND IMPROVEMENT SECTION, POMONA, CALIFORNIA

THE DISTINCTIVE NATURE OF SOME APPLE DISEASE CONDITIONS IN GEORGIA[1]

Jack Taylor[2]

Summary

Physalospora obtusa causes more losses of apples in Georgia than other fruit rotting fungi. Rots caused by Botryosphaeria ribis and Botrytis cinerea have not been serious problems. The rusts and powdery mildew have developed to only a limited extent in Georgia.

INTRODUCTION

In recent years it has become increasingly apparent that certain apple diseases may be widespread in neighboring apple-growing States and relatively unimportant in Georgia. Conversely, a disease may cause serious losses in Georgia and be less serious in nearby areas. Observations on apple black rot (caused by Physalospora obtusa (Schw.) Cke.), Botryosphaeria rot (caused by Botryosphaeria ribis (Tode ex Fr.) Gross & Dug.), Botrytis rot (caused by Botrytis cinerea Pers. ex Fr.), powdery mildew (caused by Podosphaera leucotricha (Ell & Ev.) Salm.), and cedar apple rust (caused by Gymnosporangium juniperi-virginianae Schw.) have shown that their development in Georgia has been somewhat different than in other areas. The relative unimportance of cedar apple rust can be explained on the basis of the scarcity of the alternate host, the red cedar. However, theories on some other diseases are not as plausible.

BLACK ROT

According to Anderson (2) the fruit rot phase of the apple disease caused by P. obtusa is of less importance in the eastern States than are the leaf spot and canker phases. He reports that the fungus is primarily a wound parasite and will not infect uninjured tissue, and that the rot usually appears around a wormhole or some other wound. Furthermore, it originates at the calyx end only where splitting or spray injury opens the way for infection. Others report that black rot is not of primary importance and is often a secondary invader following such fungi as B. cinerea (14). Taylor (17, 18) found that a wound apparently was not necessary for the fungus to become established in the fruit and that blossom-end rot usually originates from early season sepal infection.

In the past, workers have generally agreed on the "typical" apple black rot symptoms, but exceptions to the typical symptoms are frequent. Anderson states "In all cases the rotted apple finally turns black. The rotted tissue is somewhat firm and inclined to be rather leathery when the rot occurs before the apple is fully mature." Under Georgia conditions there is considerable variation in apple black rot symptoms depending upon variety, temperature, and so forth. The rot is usually browner and less firm on yellow apples than on red varieties.

According to Hesler (10) and Anderson (2) affected fruit was not inclined to drop and often remained on the tree for a year or more. Clayton and Aycock (5) and Taylor (17) found that the diseased fruit drop soon after the rot appears. Very few black rot mummies remain on the tree under Georgia conditions.

Originally, apple black rot was described as usually being associated with one lesion on affected fruit (10). This concept has been carried down through the years and has led to erroneous diagnoses in many cases. It is especially difficult for orchardists to separate adequately black rot from other rots, and it is important that they do so in some cases. Many Georgia growers have complained annually of failure of their spray programs to control bitter rot. Specimens from such problem orchards often proved to be black rot. Taylor (17) broadened the concept of symptomatology of apple black rot, but now it seems that the broader concept does not include the range of common symptoms, nor does it serve adequately to separate black rot from other rots.

In some cases the fruit rot caused by P. obtusa exhibits symptoms similar to those described for Botryosphaeria rot. During September 1957, about 25 percent of several hundred bushels of Red Delicious apples that had been graded and packed in perforated plastic bags were found to be rotten after being held at room temperature for about 4 days. They appeared to be disease free when graded and packed. The diseased fruit were lighter in color and softer than

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[1] Paper No. 357 of the Journal Series of the Georgia Experiment Station, Experiment, Georgia.

[2] Associate Plant Pathologist, Georgia Mountain Experiment Station, Blairsville, Georgia.

fruit affected with black rot usually are and droplets of a clear gummy liquid had formed on the surfaces. Most of the rots were diagnosed tentatively as Botryosphaeria rot on the basis of symptoms. Only about 5 percent of the apples showed symptoms typical of black rot. Platings from 100 fruit almost invariably yielded P. obtusa. The fungus was induced to sporulate in every case. B. ribis was not isolated from any fruit. Apparently, infections had occurred in the orchard and the disease had not developed sufficiently to be detected until after the fruit had been harvested, graded, and packed. Then, in the perforated plastic bags at room temperature, the rot developed rapidly. These conditions may have been conducive to development of black rot symptoms similar to those of Botryosphaeria rot.

Lewis (12) reported a core rot of apples in New York caused by B. ribis. Anderson (2) described a similar rot of undetermined internal origin caused by the same fungus. In Georgia, P. obtusa is the primary cause of core rot and apparently enters the fruit through infected flower parts (18).

The Relation of Black Rot and Frogeye Leaf Spot

The leaf spot phase of the apple disease caused by P. obtusa has a long history in Virginia (6), Pennsylvania (19), and farther north (4) as well as in more southerly areas (5, 21), but apparently the fruit rot is not as important in northern as in southern areas. In Georgia the amount of leaf spot in May is a direct indication of the amount of black rot to be expected during July, August, and September on such varieties as Detroit Red, Golden Delicious, and Red Delicious. This is because early season fruit and leaf infections occur at the same time (17). Apparently this phenomenon does not prevail in more northerly areas.

Spray tests during 1957 and 1958 on Detroit Red showed that most fruit and leaf infections occurred prior to the first cover spray in Georgia, and that wettable sulfur and liquid lime-sulfur were not effective control materials.

Inadequate pruning, sanitation practices, and spray coverage, and the use of ineffective fungicides contribute to the prevalence of black rot and leaf spot in many Georgia orchards, and probably account for much of the difference in disease severity and losses between Georgia and other eastern apple areas.

BOTRYOSPHAERIA ROT

This disease was described by Fenner (7) in 1925 and has recently received considerable attention in the East. Numerous descriptions of typical symptoms have been published, but Adams and Tamburo (1) found that the relative rarity with which B. ribis was found in fruit that exhibited the characteristic symptoms described for the disease was striking. They isolated P. obtusa from diseased apples about four times more often than B. ribis.

Botryosphaeria rot has not been a problem in Georgia and the few cases that have been identified have not been separated readily from black rot on the basis of symptoms because the range of black rot symptoms overlaps those described for Botryosphaeria rot.

In Georgia B. ribis is not found very often in dead apple twigs or cankers while P. obtusa is almost invariably found in the bark of twigs killed by fire blight or other agencies.

In almost every case where Botryosphaeria rot is a problem the Rome Beauty variety is listed as having been seriously affected (11, 15). Gallia Beauty, Golden Delicious, and Cortland (12, 20) are other varieties frequently damaged. Of these varieties, Golden Delicious is the only one that is widely planted in Georgia. This difference in varieties may account largely for the prevalence of the disease in other areas while it is relatively unimportant in Georgia.

BOTRYTIS ROT

Botrytis rot has caused some concern in Virginia and New York as a blossom-end rot. It is described as a dry rot that usually becomes corked over by harvest time on McIntosh fruit. On some other varieties, such as Delicious, a more extensive rot develops and other fungi often extend the injury until the entire fruit is rotten (13). In Virginia (14) P. obtusa is a secondary invader that extends the rot. Infection by B. cinerea occurs during May, but the disease does not manifest itself until June. Sulfur is ineffective, but ferbam and captan provide satisfactory control in Virginia and New York.

This disease has not been considered a problem in Georgia. The fungus rarely has been isolated from diseased apples and was considered to be secondary along with species of Alternaria, Rhizopus, Penicillium, and many others. The blossom-end rot occurring in Georgia is

caused by P. obtusa (17); captan offers best control and infection usually occurs during April and May.

POWDERY MILDEW

Recently powdery mildew has become a problem in the East as far south as Henderson County, North Carolina. This development of the disease seems to be associated with certain apple varieties and spraying practices (3, 14, 16). The increase in plantings of susceptible varieties such as Rome Beauty, the increased use of captan, ferbam, and glyodin instead of sulfur fungicides, and the use of concentrate sprays have probably been the primary contributing factors in the mildew problem of the East (9).

Powdery mildew has not been a problem in Georgia except in one orchard at high elevation. The grower has controlled the disease by pruning out affected branches and applying a few properly timed sulfur sprays.

DISCUSSION

In general, the differences in many apple disease problems between Georgia and other eastern apple areas seem to be primarily attributable to differences in orchard practices and predominating varieties.

Failure to renovate old orchards and establish new plantings has resulted in the accumulation of suitable substrates for many parasitic pests in Georgia. Many large apple plantings were made about 1910, and some growers have attempted to maintain commercial production in the old orchards. Many disease agents have become established in "holdover" cankers on main scaffold limbs of old trees and are a continuing source of trouble. In addition, growers often do not prune sufficiently to facilitate spraying, and do not practice satisfactory sanitation measures. Since 1950 there has been a decided increase in new plantings. Diseases should be easier to control in the young orchards.

New Georgia apple plantings show a change in varieties since 1950 (8). A greater percentage of the plantings have been Red Delicious and Rome Beauty, while Stayman Winesap, Kinnard, and Golden Delicious have shown a decrease. Red Delicious, Stayman Winesap, Golden Delicious, Detroit Red, and Rome Beauty comprised 42, 12, 8 and 0.7 percent of the trees over 10 years old, and 60, 4, 4, 8 and 12 percent of the trees less than 10 years old, respectively. This increase in planting of Rome Beauty may lead to trouble with Botryosphaeria canker and rot and powdery mildew.

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DEPARTMENT OF PLANT PATHOLOGY, GEORGIA MOUNTAIN EXPERIMENT STATION, BLAIRSVILLE, GEORGIA

SCAB CAN BE CONTROLLED ON THE SCHLEY VARIETY OF PECAN IN CENTRAL GEORGIA WITH PROPER TIMING AND THOROUGH SPRAYING

John R. Cole[1]

Although there was a heavy infection of scab, Fusicladium effusum Wint., on the foliage of both sprayed (32 percent) and unsprayed (72 percent) trees of Schley pecan in an orchard near Fort Valley, Georgia on May 15, 1958, the writer successfully controlled the disease by following the spray schedule recommended by Crops Research Division, Agricultural Research Service. This schedule called for at least six applications of bordeaux mixture, ziram or zineb.

Rainfall favored the development of scab, for 3.4 inches of rain fell on 14 days in April and 2.32 inches on 16 days in May. Furthermore, many of the rains came in late afternoon and early evening and thus the leaves and nuts remained wet all night. In addition, 7.24 inches of rain fell on 16 days in June and 8.47 inches on 20 days in July. Scab is very difficult to control under such conditions. In fact, unless a thorough job of spraying is done, continuing the spray program may be useless.

Each treatment plot consisted of a single tree replicated nine times. Trees about 40 years old required an average of 30 to 35 gallons of spray material per application. The materials were applied with a speed sprayer and the trees were circled. The treatment applied and the results obtained are given in Table 1.

Table 1. Effect of spraying Schley pecan on yield per tree and number of nuts per pound, Fort Valley, Georgia, 1958.

Spray materials	Concentration per 100 gallons	Number and description of sprays	Yield per tree (pounds)	Nuts per pound (number)
None (check)	----	----	13	105
Puratized Agricultural Spray[a] and ziram + oil[b, c]	5 pints[a] and 2 pounds + 1 pint[b, c]	2 dormant[a] + 2 prepollination[b] + 4 cover[c]	77	87
Ziram + oil[b, c]	2 pounds + 1 pint[b, c]	2 prepollination[b] + 4 cover[c]	96	78
Bordeaux mixture	4-1-100[b] and 6-2-100[c]	2 prepollination[b] + 4 cover[c]	126	78
Zineb + oil[b, c]	2 pounds + 1 pint[b, c]	2 prepollination[b] + 4 cover[c]	111	83

[a]Applied March 17 and April 3.
[b]Applied April 17 and 23.
[c]Applied May 13, June 5 and 24, and July 21.

The data in Table 1 show that all treatments gave outstanding control of scab as compared with no spray. Although the trees sprayed with bordeaux mixture produced the most nuts (pounds per tree), the quality was not any better, according to the number of nuts per pound, than that of nuts produced by trees sprayed with ziram. The data further indicate that the dormant applications of Puratized Agricultural Spray, followed by six applications of ziram, were no better than ziram alone for control of scab. The differences in yield of nuts in pounds per tree from the sprayed trees could have been caused by a variation in the set of nuts early in the season, since crop variation was observed as early as May 15.

The data in Table 1 also correspond with the scab-lesion counts made on the nuts on August 1. At that time most nuts on the check trees were severely scabbed, whereas very good control had been obtained with all spray treatments.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, ALBANY, GEORGIA

[1]Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Albany, Georgia.

A METHOD FOR MAKING RAPID PHOTOMICROGRAPHS

William J. Stone and John P. Jones[1]

Ferris and Ferris[2] suggested a rapid method of photographing nematodes by using the compound microscope as the camera and photographic enlarging paper instead of film. The prints obtained by their method were actually paper negatives and were suggested for use as records.

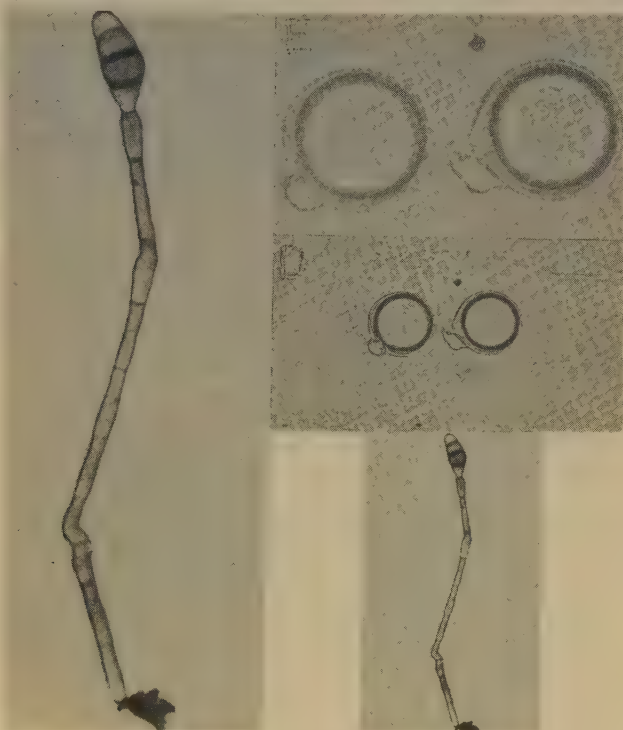

FIGURE 1.

A similar method is reported here, but Kodalith Ortho Thin Base[3], Type 2 film was used instead of enlarging paper. This film is a high-contrast film primarily designed for use in photolithography and photoengraving work. It can be developed by the same procedure and with the same developing solutions as contact prints or enlarging paper. As a result it is much faster and simpler to use than panchromatic films. This film may be handled in the dark room under yellow or red safe lights, so that it is possible to overlap many operations. The film also provides great contrast, which is desirable when hyaline conidia or mycelium is photographed. One further advantage is that much of the material photographed exhibited greater detail when water-mounted than when stained. Figure 1 shows the detail that may be obtained in contact prints and enlargements made from this type film.

A compound microscope with built-in illuminator and rheostat is used to make the picture. A device that will hold a ground glass and film holder firmly is also needed. It should hold the ground glass far enough from the microscope to give adequate enlargement, but close enough so that the operator can see the image on the ground glass and at the same time manipulate the fine adjustment of the microscope for critical focusing. A distance of 15 to 18 inches is satisfactory. A photomicrographic camera with a 5 x 7-inch ground glass and film holder has been used with very good results. However, it is not necessary to have such elaborate equipment to obtain good exposures.

The exposure time must be determined by trial and error, since the rheostat voltage, magnification and distance of the film from the microscope eyepiece all affect the time required. Once the exposure time is determined, all succeeding exposures may be made at that time as long as the previously mentioned variables remain the same and only the subject differs. For example, good negatives have been obtained when the film is exposed for 30 to 35 seconds at a distance of 16 inches from the eyepiece, using a 10X objective, a 10X eyepiece, and 8.5 volts on the rheostat. The equipment must be set up in a dark room, but yellow or red safe lights may be used while exposing Kodalith film.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND DELTA BRANCH OF THE MISSISSIPPI AGRICULTURAL EXPERIMENT STATION, STONEVILLE, MISSISSIPPI

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[1] Research Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

Appreciation is expressed to H. W. Johnson and E. R. Toole for their suggestions.

[2] Ferris, Virginia R., and J. M. Ferris. 1958. A simple method for making rapid routine photographs of nematodes. Plant Disease Reptr. 42: 1192-1193.

[3] Use of the trade name indicates a suitable type of film and does not imply recommendation of the product mentioned over other similar products.

CHEMICAL CONTROL OF ALGAE AND OTHER NUISANCE GROWTHS ON GREENHOUSE BENCHES, POTS, AND POTTING SOIL[1]

O. D. Morgan[2]

Certain algae, mosses, and fungi grow over the soil in greenhouse benches and pots, constitute a nuisance, and need to be removed from time to time. Such growths are usually controlled with steam, fumigation, or by replacing the old soil with new. However, these treatments have little or no residual effects and nuisance growths reappear soon after treatment. Since some tests with greenhouse plants require weeks or months for completion, control of these nuisance growths would be helpful in experimental work.

Copper sulfate and chlorine have commonly been used to control algae in water used for domestic and industrial purposes (1). These chemicals may, however, cause injury to certain plants and cannot be used. Recent studies (2, 3, 4, 5, 7, and 8) have indicated that large numbers of algae on ponds and in water supply reservoirs can be controlled by use of chemicals at relatively low rates when mixed with water.

In greenhouse tests where certain fungicides were applied as drenches on soil to control soil borne pathogens (6) nuisance growths were also controlled. Some of the chemicals that controlled nuisance growth were: captan (N-trichloromethylmercapto-4-cyclohexene-1,2-dicarboximide), nabam (disodium ethylene bisdithiocarbamate), Elgetol (sodium-dinitro-ortho-cresolate), copper sulfate, Puratized (phenyl-mercuri-triethano-ammonium-lactate), Isothan Q15 (2-dodecylisoquinolinium bromide), Bioquin 1 (8-hydroxyquinolin), chloranil (tetrachloro-para-benzoquinone), Mylone (3,5-dimethyl-tetra-hydro-1,3,5, 2H-thiadiazine-2-thione), the disulfide, zinc, and manganese salts of Omadine (2-pyridinethione 1-oxide), dichlone (2,3-dichloro-1,4-naphthoquinone), and chemical 5400 (alpha, alpha-trithiobis (N-dimethylthioformamide)).

Chemical 5400 and dichlone were the most effective and controlled algae, moss, and fungi for several weeks in preliminary tests. Chemical 5400 had a longer residual effect than any other chemical used. Prior to chemicals for control of nuisance growths, steam was used to treat benches, pots and soil. It was necessary to remove buried heating cables from benches before the soil could be steamed. Therefore, a study was made to determine the effectiveness of chemical 5400 and of dichlone for the control of nuisance growths on benches, pots, and potting soil.

MATERIAL AND METHODS

Material and methods used in the treatments over a 3-year period were: chemical 5400, dichlone, two 4 x 25-foot benches containing sand overgrown with algae, moss, and fungi, and one 2 x 25-foot bench used as an untreated control. About 23,000 clay pots of several sizes were used. None of the pots were washed during the 3-year period; however, once each year they were steamed to inactivate any virus that might be present in plant debris adhering to the pots. The chemicals were mixed with water and drenched on the sand and soil, and the pots were given 10 minute dips in the mixtures. Flats containing steamed soil that was overgrown with a fungus growth consisting of *Pyronema* sp., *Neurospora* sp., and *Trichoderma* sp. were treated. The algae, moss, and fungi were those commonly found in greenhouses under normal conditions. No attempt was made to identify the algae or moss. Three separate tests were run to determine the efficacy of the chemicals.

In the first test chemical 5400 was applied as a drench to sand in two large benches, 4 x 25 feet, twice each winter for 3 years. During this time the sand was not changed. A similar test with dichlone was conducted for 1 year. A 2 x 25-foot bench was used as an untreated control. Chemical 5400 was applied at the rate of 2 pounds of the 50 percent wettable powder per 100 gallons. Dichlone was applied at the same rate. Approximately 0.32 grams per square foot was applied to each large bench. All pots used in the benches during the 3-year period were dipped in chemicals at the same concentration.

The second test consisted of two replicated experiments and was run to determine the effectiveness of chemical 5400 and of dichlone on the control of algae and moss in pots. Two-

[1] Miscellaneous Publication No. 357, Contribution No. 3018 of the University of Maryland Agricultural Experiment Station, Department of Botany.

[2] Associate Professor.

inch pots and sand were steam sterilized separately. The pots were dipped in a suspension of each of the two chemicals and then filled with the sand. An algal suspension was poured over the sand in the pots. After 1 week a light green bloom of algae had overgrown the sand. At this time 25 ml of a suspension of chemical 5400 or of dichlone was poured over the sand. Four replicates were used for each treatment. Each set of four pots treated with a given concentration was placed in a plastic flat to prevent contamination, and the pots were watered as needed. Observations were made at weekly intervals to note progress of the algal growth.

A similar test was set up to determine the effectiveness of the chemicals for control of moss. Four rates of application were used: 2, 1, 1/2, and 1/4 pounds per 100 gallons of water. Each was replicated four times. A 25 ml quantity of each chemical suspension was poured onto small pads of moss cut from a bench and placed on the surface of sand in 2-inch pots. The four pots with a given rate of treatment were placed in plastic flats and watered as needed. After 6 weeks, observations were made on the degree of control of moss.

In the third test flats of steamed soil overgrown with the soil fungi, mentioned above, were treated with chemical 5400 at the rate of 2 pounds of 50 percent wettable powder per 100 gallons.

RESULTS

Results of the 3-year test indicated that chemical 5400 gave almost complete control of all nuisance growths. Figure 2 shows a part of one treated bench versus an untreated section.

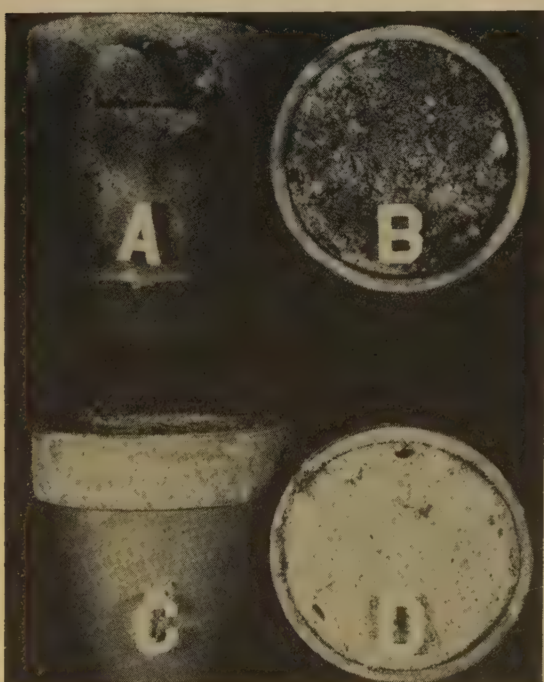

FIGURE 1. A and B, no treatment. C and D, 8 weeks after treatment with chemical 5400 at 800 ppm.

FIGURE 2. Left--no treatment. Right--8 weeks after treatment with chemical 5400 at 1200 ppm.

The two large benches were kept as clean of the growths as the treated section in Figure 2. Dichlone did not give as lasting control as did chemical 5400. The small bench used as a control was encrusted with a mat of algae and moss that had to be cleaned out each year. Neither chemical 5400 nor dichlone harmed the heating coils in the benches. Chemical 5400 caused only a light brownish discoloration of the sand, a discoloration which disappeared after watering several times. Treated pots and benches were free of algae, moss, and fungus growths for 2 1/2 to 3 months following one treatment with chemical 5400 and for 2 months after the dichlone treatment. Flats filled with steamed soil remained free of fungus growths when treated with chemical 5400, and fungus growth that was present at the time of treatment soon disappeared.

Results of the pot treatments with chemical 5400 and with dichlone to control algae and moss are shown in Tables 1 and 2 and Figures 1 and 2. Chemical 5400 at 800 and 1200 ppm controlled algae for 18 weeks, indicating a long residual effect under the conditions of the test, whereas dichlone gave partial control for 18 weeks when applied at the same concentrations (Table 1). The untreated controls had a heavy growth of algae over the sand and pots after 2 weeks (Figure 1A and B). Two pots treated with chemical 5400 at 800 ppm after 8 weeks are shown in Figure 1 C and D. Dichlone at 1200 ppm gave similar control after 8 weeks.

Table 1. The effect of chemical 5400 and of dichlone at several concentrations on the control of algae on the surface of sand in 2-inch pots[a].

Time (weeks)	Concentrations of chemicals (ppm) 50		100		200		400		800		1200		Check
	A[b]	B[b]	A	B	A	B	A	B	A	B	A	B	
1	0	0.4	0	0	0	0	0	0	0	0	0	0	3.0
2	0	1.8	0	1.0	0	T	0	0	0	0	0	0	5.0
3	T	3.1	0	2.5	0	1.0	0	T	0	0	0	0	5.0
4	1.0	4.4	T	3.3	0	2.6	0	0.8	0	0	0	0	5.0
5	2.4	5.0	1.9	4.0	0.8	4.3	0	1.6	0	0.5	0	T	5.0
6	4.3	5.0	3.8	4.0	1.5	5.0	T	3.8	0	2.3	0	1.5	5.0
18	5.0	5.0	5.0	5.0	4.0	5.0	1.0	5.0	0	3.6	0	2.2	5.0

[a] Index increments were 0 = none, T = trace, 1 = 20 percent of sand covered, 5 = 100 percent of sand covered with a heavy growth of algae.
[b] A = Chemical 5400. B = dichlone.

Table 2. The effect of chemical 5400 and of dichlone at several concentrations on control of moss on greenhouse pots.

Rate (pounds per 100 gallons)	Percentage of moss killed after 6 weeks: Chemical 5400	Dichlone
2	86.3	32.5
1	50.0	0
1/2	20.0	0
1/4	7.0	0
None	0	0

Control of moss was not as complete as control of algae (Table 2). Six weeks after treatment with chemical 5400 or with dichlone, each at the rate of 2 pounds of the 50 percent wettable powder per 100 gallons of water, growth of the moss was controlled to the extent of 86 percent and 32.5 percent, respectively. Estimate of moss growth 6 weeks after treatment showed that, at the 1 pound per 100 gallon rate, chemical 5400 gave 50 percent control as compared with no control with dichlone at the same rate. After 6 weeks the moss started to grow over the pots again. Moss can be controlled effectively by chemical 5400 if it is applied to the soil before the moss becomes established.

Control of algae and moss has also resulted in the elimination of slug damage; even when these pests are introduced from other sections of the greenhouse they soon disappear.

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DEPARTMENT OF BOTANY, UNIVERSITY OF MARYLAND AGRICULTURAL EXPERIMENT STATION, COLLEGE PARK

FIRST REPORT OF MELOIDOGYNE JAVANICA ON GREENHOUSE GROWN SOLANUM PSEUDO-CAPSICUM[1]

D. P. Taylor[2]

Several authors have cited Jerusalem or Christmas cherry, Solanum pseudo-capsicum, as a host for root-knot nematodes; however, in no case was the species of Meloidogyne mentioned. This disease of Jerusalem cherry was first reported by Gardner (3) as being caused by Heterodera radicicola (Greef, 1872) Mueller, 1884. Root knot has also been reported on this ornamental by Barss (1) as caused by Caconema radicicola (Greef, 1872) Cobb, 1924; Buhrer et al. (2) and Weiss (5) as caused by Heterodera marioni (Cornu, 1879) Goodey, 1932; and Minz (4) as caused by Meloidogyne sp.

FIGURE 1. Root system of S. pseudo-capsicum with root-knot galls caused by M. javanica.

Jerusalem cherries grown in a St. Paul conservatory exhibited sparse foliage, small leaves, and few flowers and berries. Examination of the root systems of these plants disclosed the presence of numerous root-knot galls (Figure 1). On the basis of perineal patterns, specimens dissected from galls were identified as Meloidogyne javanica (Treub, 1885) Chitwood, 1949. Impatiens sultani growing in the same planting were also heavily attacked by this species although growth was not visibly affected. This constitutes a first report of M. javanica on S. pseudo-capsicum and a first report of root knot on Jerusalem cherry in a greenhouse in Minnesota.

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DEPARTMENT OF PLANT PATHOLOGY AND BOTANY, INSTITUTE OF AGRICULTURE, UNIVERSITY OF MINNESOTA, ST. PAUL, MINNESOTA

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[1] Paper No. 4111, Scientific Journal Series, Minnesota Agricultural Experiment Station.

[2] Instructor, Department of Plant Pathology and Botany, University of Minnesota.

AN EVALUATION OF ROLE OF DINOSEB IN "NON-DIRTING" CONTROL FOR PEANUT STEM ROT

Kenneth H. Garren

In 1958 Garren and Duke (4) reported that for 3 years, under Virginia conditions, the deep covering of organic matter on land prior to planting peanuts (Arachis hypogaea), followed by "non-dirting" cultivation that avoided bringing soil in contact with the plants, effectively controlled the stem rot caused by Sclerotium rolfsii Sacc. Early in the investigation attention was directed to two reports suggesting the possible fungicidal role of the dinoseb[1] used as a pre-emergence herbicide in the non-dirting cultivation.

The first of these was the 1956 report of Chappell and Miller (2) whose laboratory work showed the growth of S. rolfsii was greatly retarded when inoculated on culture media containing field rate concentrations of certain herbicides including dinoseb. They recorded the incidence of stem rot on four farms where dinoseb was used pre-emergence at 9 pounds per acre, in terms of dead plants showing infection by S. rolfsii per 300 feet of row. The averages were 3.2 plants for the checks and 1.3 for dinoseb-treated areas. Chappell and Miller did not establish that the herbicides actually acted as fungicides, but they concluded that the herbicides tested may influence disease development in peanuts. In 1956 Campbell (1) reported that numerous experiments showed that dinoseb and certain fungicides used as pre-emergence soil-surface sprays were effective in controlling mint rust.

Consequently, a split-plot experiment was set up at Holland, Virginia in 1956. In this experiment land preparation was conventional and the main plots were dirting and non-dirting cultivation as described by Garren and Duke (3). The sub-plots were dinoseb and no dinoseb. The dinoseb was applied pre-emergence at a rate found to give adequate weed control, that is, 3 gallons of 53 percent active substance per acre. Dixie Spanish variety peanuts were used in 1956 and Virginia Bunch variety in 1957 and 1958. Soon after emergence, plantings were spot thinned to comparable stands for all plots each year. Plots were examined every week and plants were tagged as soon as infection by S. rolfsii was evident.

In 2 years out of 3 there was a significant decrease in infection associated with use of dinoseb (Table 1). Also in 2 years out of 3 there was a significant increase in yield associated with use of dinoseb. However, the significant reduction in infection and the significant increase in yield coincided only in 1956. Average reduction in infection from use of dinoseb was 35 percent; average increase in yield was 7.8 percent.

Table 1. Prevalence of Sclerotium rolfsii infection on bunch peanuts and pod yields, 1956-1958. Holland, Virginia.

Treatment	Stand infected (percent)				Pod yield per acre (pounds)			
	1956	1957	1958	Average	1956	1957	1958	Average
Herbicide								
Dinoseb	12.0	7.2	15.8	11.7	1806	2663	2995	2488
No dinoseb	17.8*	16.1**	20.0	18.0	1589*	2610	2738**	2309
Cultivation								
Dirting	21.6	15.0	28.6	21.7	1309	2712	2584	2202
Non-dirting	7.3**	8.6*	7.2**	7.7	2086**	2560	3149**	2598

** Significant at the 1% level.
* Significant at the 5% level.

1 Dinitro-o-secondary butyl phenol. "Dinoseb" is the common name for this chemical as recognized by the Association of American Pesticide Control Officials in their "Pesticide Chemicals Official Compendium."

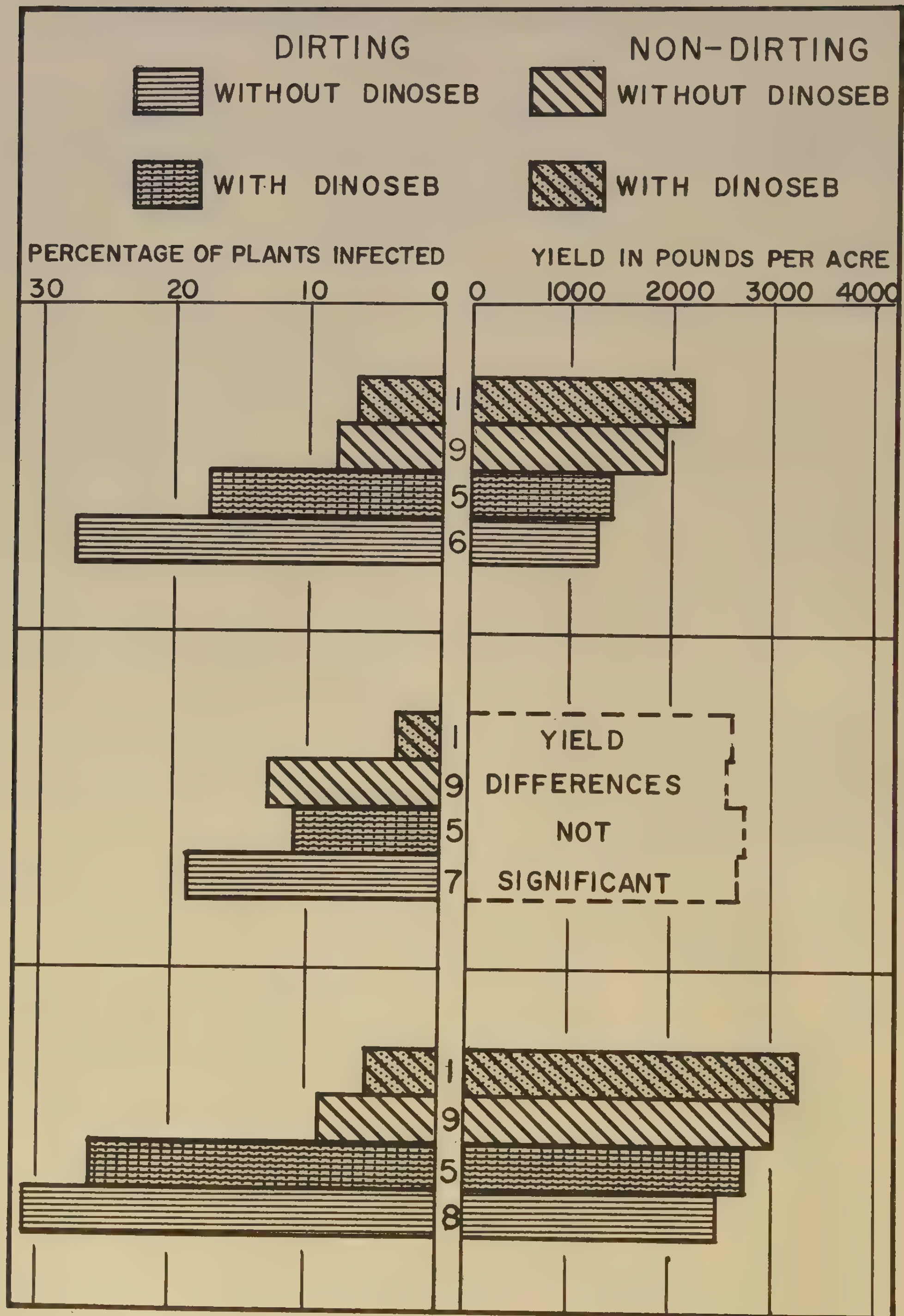

FIGURE 1. Relation of infection by Sclerotium rolfsii to yields of bunch peanuts for two cultivation treatments with and without dinoseb in 3 years. Holland, Virginia.

In contrast, non-dirting cultivation significantly reduced the proportion of plants infected every year and highly significantly increased yield in 2 years out of 3, as compared with dirting cultivation (Table 1 and Fig. 1). The average reduction in infection from use of non-dirting cultivation was 65 percent; the average increase in yield was 18 percent. Non-dirting cultivation was nearly twice as effective in reducing infection and more than twice as effective in increasing yield as was dinoseb. The average infection level when dinoseb was used was 52 percent higher than that when non-dirting cultivation was used. Interaction between herbicide and cultivation was not statistically significant for plants infected or for yield in any test.

Although dinoseb, as applied, was somewhat toxic to S. rolfsii, the toxicity was not enough to justify recommending the use of dinoseb as a control measure for peanut stem rot. The use of dinoseb in non-dirting cultivation must be justified primarily on the basis of its control of weeds.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND TIDEWATER RESEARCH STATION OF VIRGINIA EXPERIMENT STATION, HOLLAND, VIRGINIA

CERCOSPORA THEAE ON CAMELLIA SASANQUA AND THEA SINENSIS

A. G. Plakidas

On February 5, 1958 one of the State nursery inspectors brought to us three small potted diseased Camellia sasanqua Thunb. plants for diagnosis. The foliage was covered with numerous purplish spots, the older ones with necrotic grayish centers. No fungus fructifications were discernible to the naked eye, but examination with the dissecting microscope showed numerous black specks dotting the upper surface of the older, necrotic lesions. These proved to be dense stromatic structures, dark brown in color, and situated subcuticularly to subepidermally. No conidia were found; however, when the plants were placed in a moist chamber overnight, some of the stromata gave rise to clusters of thin, cylindric, hyaline, septate, conidia ranging in length from about 35μ to 85μ. Both the pathogen and the disease were unknown to the writer.

Three weeks later, the nursery in which the original specimen was collected (in Loranger, Louisiana) was visited. The disease was found in severe form on several thousand small potted C. sasanqua plants growing very close together in one section of a large lath house. The foliage was covered with very numerous spots, there was considerable defoliation and dieback of twigs (Figs. 1 and 2), and many plants were dead. The disease was limited to two varieties, Texas Star and Pink Snow. Other varieties, adjacent to the diseased ones, were completely free of the disease. The owner stated that he had first noticed the disease the previous September in a small area among the Texas Star plants and from there it spread gradually during the fall and winter months.

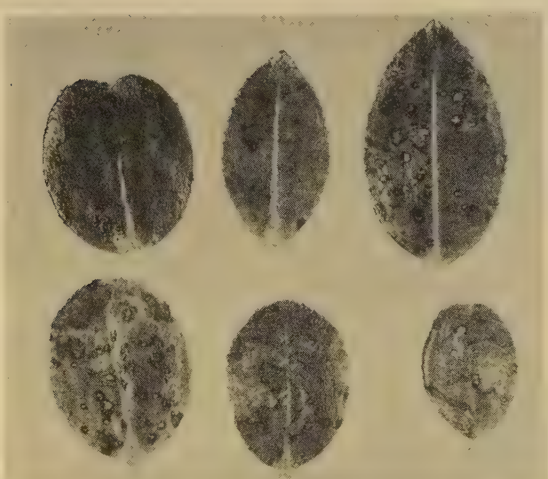

FIGURE 1. Typical lesions on Camellia sasanqua leaves.

FIGURE 2. Defoliation and dieback of Camellia sasanqua var. Texas Star.

IDENTITY OF THE PATHOGEN

Because the fruiting bodies were noted to arise subcuticularly, they were believed to be acervuli, and the fungus was tentatively identified as a Cylindrosporium. However, it was also appreciated that the fungus could perhaps fit as well in the genus Cercospora. Dr. Charles Chupp of Cornell University kindly examined specimens submitted to him and identified the fungus as Cercospora theae (Cav.) B. de Haan. Following Dr. Chupp's identification, the same fungus was found on spotted leaves of four potted tea (Thea sinensis L.) plants growing in a lath house in Baton Rouge. The lesions on tea (Fig. 3) were much larger than those on C. sasanqua.

FIGURE 3. Typical lesions on leaves of tea (Thea sinensis).

The known distribution of Cercospora theae, according to Chupp (A monograph of the fungus genus Cercospora, Ithaca, New York, 1953, p. 561) is Java, Caucasus, Ceylon, Formosa, Japan, Italy, and Mauritius. The only reference to Cercospora on camellia in this country is the listing, "Cercospora sp., leaf spot. Ga." (Index of Plant Diseases in United States," p. 1138). This listing, according to the records of Mycology and Plant Disease Reporting Section, United States Department of Agriculture, is based on a single camellia leaf specimen in the herbarium of the National Fungus Collections. The specimen was collected by a Plant Quarantine inspector (Blizzard) at Richmond Hill, Georgia, December 11, 1943. The determination was made by D. P. Limber, with a notation on the packet, "We have no record of a Cercospora on this host."

The surviving diseased C. sasanqua plants were transplanted in the field in March, 1958. They were examined periodically during the summer to check on the progress of the disease. The plants grew well and only very light infection occurred on the new foliage. A check was also made of C. sasanqua plants of several varieties growing in the field in two separate nurseries in the vicinity. The disease was found on plants of the same two varieties, Texas Star and Pink Snow, but infection was very light; only a few scattered lesions were present. It would seem, then, that this disease is capable of causing serious damage only under environmental conditions that favor its development, such as the crowding, partial shade, and high humidity of a lath house.

LOUISIANA STATE UNIVERSITY, BATON ROUGE

BLIGHT OF MARIGOLD, TAGETES ERECTA, CAUSED BY COLLETOTRICHUM CAPSICI (SYD.) BUTL. & BISBY

H. K. Saksena and B. B. Singh[1]

Abstract

A new leaf, inflorescence and stem blight of Tagetes erecta (African marigold) caused by Colletotrichum capsici (Syd.) Butl. & Bisby is described. Cross inoculation studies have shown the isolate of Colletotrichum from marigold to cause fruit rot of chillies (Capsicum annuum) and C. capsici, causal organism of dieback of chillies, to attack marigold. Preliminary experiments on control of disease have shown the efficacy of Bordeaux mixture and zineb in reducing the disease incidence.

INTRODUCTION

The african type marigold, Tagetes erecta, is universally grown as a garden plant for the decorative value of its flowers. It is an important cash crop in places of religious importance in India. A severe blight disease of marigold was observed in the month of July 1956 in the botanical garden of the Government Agricultural College, Kanpur, India. A limited survey of the vicinity of Kanpur showed the disease to be present in most of the gardens visited, shortening the life of marigold as a flowering ornamental. Repeated isolations from diseased parts of plants always produced cultures of a Colletotrichum sp.

Weiss (8) listed many parasites causing diseases of marigolds. Recently Edward (3), from India, and Changsri and Weber (1), from the United States, described Alternaria zinniae and Septoria tageticola, respectively, causing diseases of marigolds. Since no report of Colletotrichum spp. on Tagetes could be found in literature, the present studies on the disease were undertaken.

SYMPTOMATOLOGY

The disease first manifests itself as grey spots of variable shape and size on the involucral bracts and leaflets, which enlarge rapidly, coalesce and become water-soaked. Under favorable conditions, the wet rot progresses downward to peduncles and petioles and finally to branches, killing the infected tissues. The affected flower buds fail to open and droop down due to necrosis and collapse of the cells of peduncles. The individual flowers in the involucre show rotting to start at the point of their attachment to capitulum. As the disease progresses, the leaflets become curled and twisted and some are shed. The branches show girdling at the site of infection. At advanced stage of disease development, the infected parts shrivel, dry and turn black (Figure 1). Such plants present a parched appearance. Numerous black dot-like acervuli are formed irregularly scattered or in small groups at places over the infected surface. The conidia from these continue to spread the secondary infection rapidly.

Plants in any stage of growth were susceptible to infection. The blight was observed to cause early death of most of the affected plants. Severe outbreaks depended mostly on continued warm and humid weather conditions which quite frequently prevail during monsoon season. In fields, the diseased plants were observed in scattered groups. Plants grown before July usually seem to escape the disease because of dry weather.

EXPERIMENTS AND RESULTS

Throughout the study pure cultures of fungus obtained from naturally infected marigold plants were used. Colletotrichum capsici, causal organism of dieback of chillies (Capsicum annuum), which is the most common species occurring in India and elsewhere on a wide variety of hosts, was also isolated for comparative morphological and inoculation studies. Pathogenicity tests were made on plants of T. erecta grown in pots in partially sterilized soil. These were sprayed with a thick suspension of conidia, incubated in moist chambers for 48 hours and

[1] Assistant Professor of Plant Pathology and Research Assistant, respectively, Government Agricultural College, Kanpur, India.

FIGURE 1. Symptoms of blight on marigold inoculated with Colletotrichum capsici.

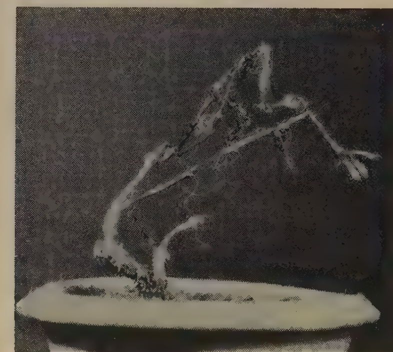

FIGURE 2. Marigold plant killed at advanced stage of blight development.

then removed to greenhouse benches.

The Pathogen

The marigold isolates of Colletotrichum grew well on potato-dextrose, Richard's, and oatmeal agar media. The aerial growth was fluffy and mouse grey in color. Mycelial growth was composed of both hyaline and brown hyphae, the latter forming chains of chlamydospores. The hyphae measured 3.6 - 8.5μ in width. The average size range of acervuli, setae, conidia and conidiophores of Colletotrichum isolate from marigold and C. capsici from chillies on PDA after 20 days of growth and on their respective host plants is shown in Table 1. Fifty measurements each of the above structures were made for each isolate, including measurements for each substrate on which the isolate fructified.

Table 1. Measurements (μ) of acervuli, setae, conidia and conidiophores for the two isolates of Colletotrichum.

Isolate and substrate	Acervuli	Setae	Conidia	Conidiophores
Marigold				
Host	69.2-164	82.0-278.8 x 3.6-7.3	20.1-27.6 x 1.8-3.6	12.8-25.2 x 2.5-4.6
Culture	82.0-180	98.6-205.0 x 2.8-6.4	16.0-27.2 x 1.8-3.6	21.9-29.2 x 2.7-4.8
Chillies				
Host	106.0-229	56.7-173.8 x 5.5-7.3	18.3-25.6 x 2.7-3.8	16.4-27.4 x 2.7-4.1
Culture	98.4-205	73.8-205 x 3.6-6.4	16.4-27.4 x 2.7-3.6	23.7-30.6 x 2.5-4.6

The acervuli usually took longer (12 to 15 days) to come up in culture than on host plants. They are circular, erumpent, dark brown to black. The almost black and gradually tapering setae are irregularly scattered all over the surface of the acervulus. They are septate with

2 to 7 septa and their number per acervulus varied from 20 to 85.

The conidiophores are short, hyaline, simple erect and crowded in layers, and vary in shape and size. The conidia, produced in pinkish or creamy masses, are unicellular, hyaline, falcate and bluntly tapering at both ends with single or two oil globules in the center.

Pathogenicity

All the 15 plants of T. erecta inoculated with blight organism developed symptoms on the leaflets and flower buds on the third day. Further development of disease was rapid and affected parts were profusely covered with white mycelium which persisted for 2 to 3 days. Acervuli development was observed on the tenth day and 25 days after inoculation the affected plants were killed (Figure 2). C. capsici from chillies caused early symptoms of disease on the other set of T. erecta plants inoculated with it and the infection was restricted to flower buds and young leaflets.

In further cross inoculation tests, the marigold isolate of Colletotrichum caused typical symptoms of dieback on 12 out of 25 inoculated fruits of chillies. Generally the initial symptoms (Figure 3) and the development of black acervuli arranged in concentric rings was similar to those produced by C. capsici; however, the severity of disease and the number of inoculated fruits affected was greater in the latter (21 out of 25).

FIGURE 3. Dieback of fruits of Capsicum annuum caused by Colletotrichum capsici isolates obtained from (left) chillies, (right) marigold, (center) check.

CONCLUSIONS

The causal agent of blight of T. erecta possesses a thick stroma, setae and falcate unseptate spores. These characters place the fungus under the genus Colletotrichum Corda. The acervuli of marigold isolate of Colletotrichum vary greatly in size from those of C. capsici isolate. Previous workers (2, 4, 5, 6) found similar variations in the size of acervuli and setae and the number of setae per acervulus of Colletotrichum and it was suggested that structures that exhibit such wide variations can hardly be relied upon for specific differentiation. The shape and size of conidia have been commonly utilized as reliable characters. The size measurements of conidia of marigold isolate are in agreement with that described for C. capsici and also conform with the size of conidia of the isolate of the latter used in these studies. Cross inoculation studies have shown the marigold isolate to infect fruits of chillies and C. capsici from chillies to attack marigold. The causal organism of blight disease of T. erecta is, therefore, identified as C. capsici. This is considered to be the first record of the fungus on this host. C. capsici is known to parasitize a great variety of plants. Detailed comparative studies by Ramakrishnan (6, 7) have shown the isolates of Colletotrichum obtained from cabbage, knol-khol, cauliflower, Colletotrichum curcumae on turmeric, C. indicum on cotton and C. truncatum to differ in no essential way from C. capsici and according to him they should all be merged in one species.

Infection is mainly airborne. Preliminary experiments carried out have shown that 1 percent Bordeaux mixture and 2 percent zineb, sprayed on or before the commencement of disease and repeated at 14-day intervals, keeps the infection well down.

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SECTION OF BOTANY AND PLANT PATHOLOGY, GOVERNMENT AGRICULTURAL COLLEGE, KANPUR, INDIA

BRIEF NOTE ON PLANT DISEASE OCCURRENCE

TAR SPOT OF CORN IN GUATEMALA

By Eugenio Schieber

Tar spot of corn, incited by Phyllachora maydis Maubl., was found severely attacking indian corn in the highlands of Guatemala, especially in the Departamentos of Chimaltenango and Solola at an elevation of 6000 feet above sea level. A specimen collected in September 1958, near Agua Escondida, Solola was identified as Phyllachora maydis Maubl., and confirmed by Drs. M. P. Backus and H. C. Greene of the Botany Department, University of Wisconsin, Madison, Wisconsin. Apparently the first report of this pathogen from Guatemala was given by I. E. Melhus[1] as causing little injury. This appears to be the first report of economic importance for Guatemala. In Central America it has also been reported from Nicaragua in the preliminary list of Nicaraguan plant diseases (Plant Disease Reptr. Suppl. 243). Stevenson[2] reported it from Puerto Rico, Cuba and Mexico.

PLANT PATHOLOGY DEPARTMENT, UNIVERSITY OF WISCONSIN, MADISON, WISCONSIN

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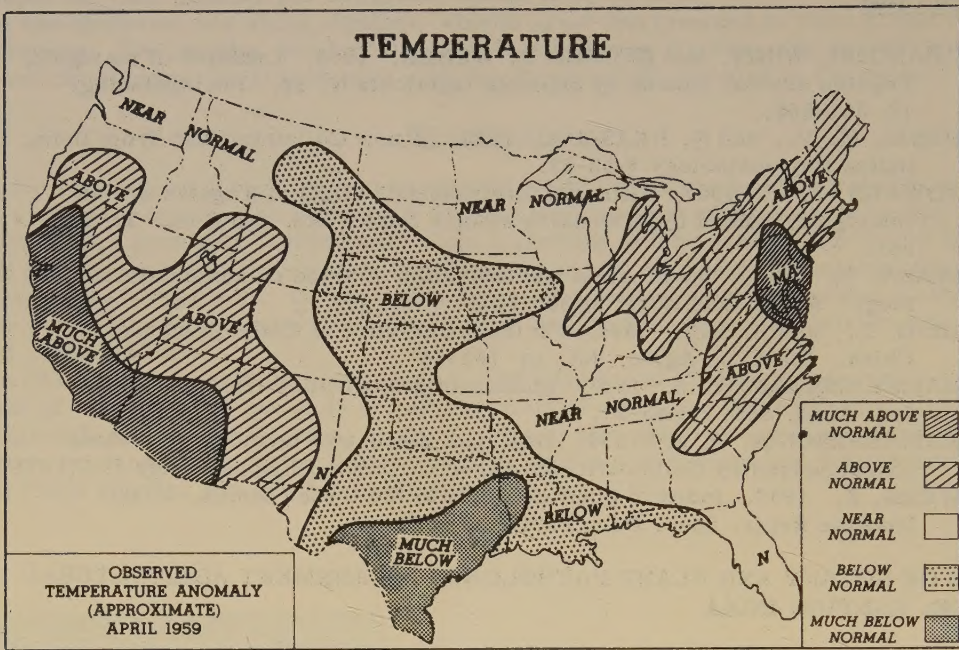

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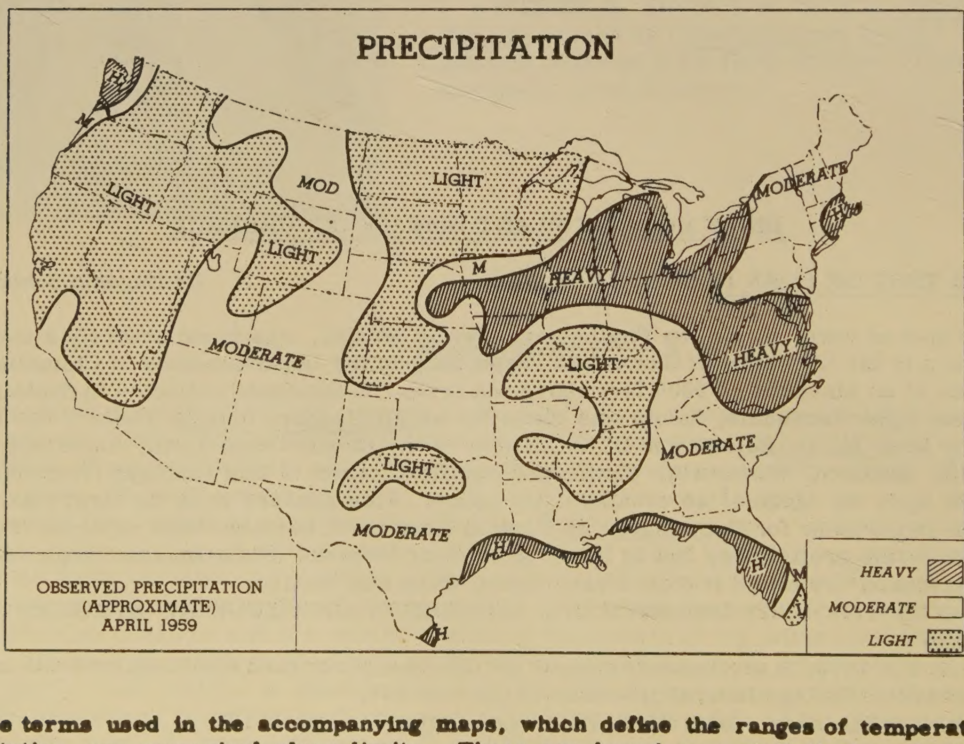

OBSERVED PRECIPITATION (APPROXIMATE) APRIL 1959

The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.